P
Med
A

TRANSACTIONS

OF THE

THIRTEENTH ANNUAL MEETING

OF THE

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

HELD IN

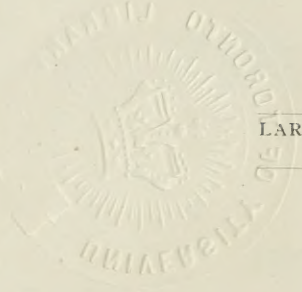
NEW YORK CITY,

May 30, 31, and June 1, 1907.

370731
28.8.39

St. Louis:
Published by the Society.
1907.

LARYNGOSCOPE PRESS
SAINT LOUIS



RF
1
A53
1907

OFFICERS

President.

WENDELL C. PHILLIPS, New York, N. Y.

Vice-Presidents.

Eastern Section:
FRANK B. SPRAGUE, Providence, R. I.

Middle Section:
J. M. INGERSOLL, Cleveland, Ohio.

Western Section:
W. C. BANE, Denver, Colo.

Southern Section.
J. M. RAY, Louisville, Ky.

Secretary.

THOMAS J. HARRIS, 117 East 40th St., New York, N. Y.

Treasurer.

EWING W. DAY, Westinghouse, Bldg., Pittsburg, Pa.

Council.

JAMES E. LOGAN, Kansas City, Mo.
FREDERICK C. COBB, Boston, Mass.
NORVAL H. PIERCE, Chicago, Ill.
LEWIS A. COFFIN, New York, N. Y.
THOMAS H. HALSTEAD, Syracuse, N. Y.
JAMES F. McKERNON, New York, N. Y.
H. W. LOEB, St. Louis, Mo.
CHARLES W. RICHARDSON, Washington, D. C.
D. BRADEN KYLE, Philadelphia, Pa.

Library Committee.

H. HOLBROOK CURTIS, New York, N. Y.
W. SOHIER BRYANT, New York, N. Y.
DAVID G. YATES, New York, N. Y.
W. P. BRANDEGEE, New York, N. Y.
P. D. KERRISON, New York, N. Y.

Publication Committee.

CHEVALIER JACKSON, Pittsburg, Pa.
W. H. HASKINS, New York, N. Y.
JOHN D. RICHARDS, New York, N. Y.

LIST OF PRESIDENTS

1896. EDWARD B. DENCH, M. D.
1897. FRANK HYATT, M. D.
1898. WILLIAM H. DALY, M. D.
1899. S. E. SOLLY, M. D.
1900. D. BRADEN KYLE, M. D.
1901. ROBERT CUNNINGHAM MYLES, M. D.
1902. CHARLES W. RICHARDSON, M. D.
1903. J. A. STUCKY, M. D.
1904. NORVAL H. PIERCE, M. D.
1905. FREDERIC C. COBB, M. D.
1906. JAMES E. LOGAN, M. D.
1907. WENDELL C. PHILLIPS, M. D.

TABLE OF CONTENTS.

PAGE

Address of Welcome, JOHN A. WYETH, M. D. 1

President's Annual Address, WENDELL C. PHILLIPS, M. D. 2

Papers.

Modern Methods in The Repair of Cleft Palate. JOHN B. ROBERTS, A. M., M. D. 10

Operations for Cleft Palate and Their Results, Especially in Respect to the Improvements of Speech. G. HUDSON-MAKUEN, M. D. 15

The Premaxillary Wings and Deviations of the Septum. HARRIS PEYTON MOSHER, M. D. 26

A Report of a Case of Fibro-Myxoma, Involving the Left Superior Maxilla, Including the Alveolus, Floor, Anterior and Posterior Walls, and of Seven Years' Duration. W. H. HASKIN, M. D. 57

The Treatment of Foreign Bodies in the Respiratory Tract and Esophagus. Professor GUSTAV KILLIAN 63

The Questionable Influence of So-Called Diathetic Conditions in Diseases of Throat and Nose in Children. CHARLES G. KERLEY, M. D. 101

Hysteria of the Ear. CHRISTIAN R. HOLMES, M. D. 107

A Study of Ear Symptoms in Arterio-Sclerosis, with Special Reference to the Labyrinth. JOHN J. KYLE, M. D. 123

Visual Disturbances Showing a Causal Relation to Disease in the Sphenoidal Sinuses, with the Report of a Case, Together with Coronal and Sagittal Sections, Demonstrating the Relations Existing Between These Cavities. JOHN W. MURPHY, A. M., M. D. 135

Symposium—Diseases of the Labyrinth.

Purulent Affection of the Labyrinth Consecutive to Disease of the Middle Ear. Pathology. CLARENCE JOHN BLAKE, M. D. 142

The Symptomatology and Diagnosis of Labyrinthitis, Consecutive to Purulent Otitis Media. H. O. REIK, M. D. 152

Surgery of the Labyrinth. JOHN D. RICHARDS, M. D. 161

Symposium—The Faucial Tonsil.

Pathology of the Faucial Tonsil. JAMES A. BABBITT, M. D. 189

Modern Surgery of the Faucial Tonsils. ROBERT C. MYLES, M. D. 201

Surgery of the Faucial Tonsil. THOMAS J. GALLAHER, A. M., M. D. 206

PAGE

The Suppurating Middle Ear Diseases of Infancy and Childhood. S. J. Kopetzky, M. D. 216

Symposium—Tuberculosis.

Tuberculosis of the Mouth. Robert Levy, M. D. 253

The Etiology of Tuberculosis of the Upper Respiratory Tract. George Bacon Wood, M. D. 264

The Baneful Influence of Pregnancy on Laryngeal Tuberculosis. Wolff Freudenthal, M. D. 274

A Certain Phase in the Differential Diagnosis of Tuberculosis and Syphilis of the Upper Respiratory Tract. Lee Maidment Hurd, M. D. 283

A Case of Primary Carcinoma of the Uvula. Operation. No Recurrence. Clement F. Theisen, M. D. 289

A Second Report of a Case of Epithelioma of Uvula, Operated Five Years Ago Without Recurrence. James F. McCaw, M. D. 295

Observations on the Temporal Bone. R. Johnson Held, M. D. 297

Presentation of Cases.

Adeno-Carcinoma of the Maxillary Antrum. Lee M. Hurd, M. D. 311

Primary Tuberculosis of the Septum. Joseph H. Abraham, M. D. 311

Edematous Nasal Polyp. Wendell C. Phillips, M. D. 311

Angioma. Wendell C. Phillips, M. D. 312

Two Cases of Otitis Media Catarrhalis Chronica Showing Improved Hearing After Acute Mastoiditis Treated by Operation. William Sohier Bryant, M. D. 313

Cleft Palate. Truman M. Brophy, M. D. 313

Laryngeal Tuberculosis. Robert Levy, M. D. 313

A Case of Angio-Neurotic Edema of the Throat. Thomas H. Halsted, M. D. 314

Presentation of Instruments.

Safety Pin Closer. Chevalier Jackson, M. D. 314

Anti-Tubercle Screen. Robert Levy, M. D. 315

A Snare. L. L. Mial, M. D. 315

Presentation of Pathological Exhibit.

Anomalies of Accessory Sinuses 317

Tubercular Tumor of the Nasal Septum. Joseph H. Abraham, M. D. 318

Laryngeal Tumor. Tumor of the Middle Turbinate. Fibro-Myxomo of the Upper Jaw. E. G. Zabriskie, M. D. 318

Sarcoma of the Antrum. J. Price Brown, M. D. 319

MEETING OF EASTERN SECTION.

Papers.

PAGE

A Case of Sarcoma of the Maxillary Sinus. F. T. Rogers, M. D. 320

Professor Gustav Killian and His Work. Experiences of a Recent Visit to Freiburg, Germany. William F. Chenery, M. D. 323

Thymic Tracheo-Stenosis. Tracheoscopy. Thymectomy. Cure. Chevalier Jackson, M. D. 328

An Unusual Cyst of the Larynx. D. Crosby Greene, Jr., M. D. 338

A Case of Chronic Sinusitis, Cured by Intra-Nasal Treatment. J. Payson Clark, M. D. 343

Three Cases of Labyrinthine Suppuration Complicating Purulent Otitis Media. Edward Bradford Dench, M. D. 348

A Derma-Periosteal Flap in Radical Mastoid. Edgar M. Holmes, M. D. .. 357

Report of a case of Cerebellar Abscess. Operation. Recovery. John D. Richards, M. D. 358

Report of Three Interesting Cases. T. J. Harris, M. D. 366

MEETING OF MIDDLE SECTION.

Papers.

Upper Tracheoscopy and Bronchoscopy. Chevalier Jackson, M. D. 372

Report of Case of Traumatic Ethmoiditis. Cerebral Abscess. Death During Operation. Autopsy. J. A. Stucky, M. D. 376

Indication for Radical Operation on the Nasal Accessory Sinuses. Norval H. Pierce, M. D. 379

Concerning the Radical Treatment of Chronic Diseases of the Antrum, with Suggestions for a New Method in Operating by the Submucous Resection of the Lateral Nasal Wall. R. Bishop Canfield, M. D. 384

The Acoustics of the Mouth and the Relation of the Individual's Voice to Hearing. D. Braden Kyle, A. M., M. D. 394

Two Cases of Extensive Cholesterin Infiltration of the Mastoid Cells. Edward Bradford Dench, M. D. 401

Atrophic Rhinitis. Etiology and Treatment. Illustrated by Radiographs. Joseph C. Beck, M. D. 405

The Importance of Correcting Pathological Conditions of the Nose and Throat in Patients Who Have Incipient Tuberculosis. Willis S. Anderson, M. D. 417

Further Studies in Nasal Therapeutics. John A. Thompson, M. D. 423

Two Chisels Designed for the Prevention of Injury to the Facial Nerve in the Radical Mastoid Operation. John J. Kyle, M. D. 429

Primary Melano-Sarcoma of the Nose. Report of a Case and Review of the Literature. Cornelius G. Coakley, M. D. 430

Hypertrophy of Pharyngeal Lymphatic Ring as a Causative Factor in the Production of Epileptic Equivalents. Walter S. Daly, M. D. 441

MEETING OF THE SOUTHERN SECTION.

Papers. PAGE

When Shall We Advise Tympano-Mastoid Exenteration in the Treatment of Suppurative Otitis Media, and in What Percentage of Cases May We Expect a Cure. H. O. Reik, M. D. 448

Treatment of Atrophic Rhinitis with Strong Solutions of Nitrate of Silver with Massage. George F. Keiper, M. D. 459

Primary Syphilis of the Lips and Tonsils. A. R. Thrasher, A. M., M. D. 462

A Study of the Etiology of Inflammatory Diseases of Nose and Accessory Sinuses. William Lincoln Ballenger, M. D. 466

MEETING OF THE WESTERN SECTION.

Papers.

Exhibition of Cases 482

The Antrum of Highmore as an Etiological Factor in the Production of Hay Fever (So-Called). Jacob E. Schadle, M. D. 484

On the Obliteration of the Accessory Nasal Sinuses. A. R. Solenberger, M. D. 511

Tooth Cysts. Thomas J. Gallaher, A. M., M. D. 520

A Treatment of Acute Otitis Media. P. F. Gildea, M. D. 523

Ocular Diseases of Nasal Origin. James Allen Patterson, M. D. 524

In Memoriam 530

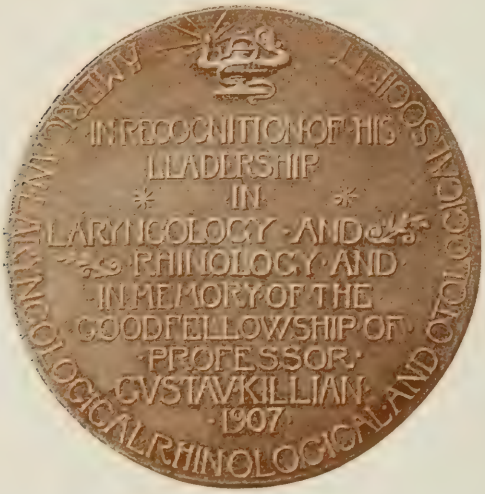
Business Meeting 533

Report of the Secretary 539

Fellows 545

Honorary Fellows 551

FAC-SIMILE OF THE BRONZE MEDALLION VOTED BY THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY IN COMMEMORATION OF THE VISIT OF PROFESSOR GUSTAV KILLIAN AS ITS GUEST IN JUNE, 1907.



TRANSACTIONS
OF THE
THIRTEENTH ANNUAL MEETING
OF THE
AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

Held in New York City, May 30, 31 and June 1, 1907.

ADDRESS OF WELCOME.

JOHN A. WYETH, M. D.
President of the N. Y. Academy of Medicine.

Gentlemen of the American Laryngological, Rhinological and Otological Society, Welcome:

We of the Academy respect you, not only because you are *numerous* and *vigorous,* but because you are *clever.* Our own Greater New York, and our Academy is full of you. You are everywhere, in everything, especially *in pocket.* The author of "Hudibras," with prophetic vision, must have had you in mind when he wrote:

"The turnpike road to people's hearts I find,
Leads through their mouths, or I mistake mankind."

Wiser in your generation, you have discounted the poet in so many ways. He perished for want of bread. It was of him that Thomas Hood wrote, upon the occasion of the unveiling of a monument:

"While Butler, wretched man, was yet alive,
No generous patron would a dinner give;
But see him dead, his bones resolved to dust,
Presented with a monumental bust.
The poet's fate is here in emblem shown,
He asked for bread, and he received a stone."

Not so with you. No laryngologist, rhinologist or otologist ever starved to death in this country, unless he had a stricture of the oesophagus, and that very low down.

Beside the mouth, you have found two other ways of reaching the hearts of men. When this opening is barred, you not only

climb in through the window of the nose, but you crawl in through the back-door of the ear, and so, one way or another, you reach the great warm centre of *being*, and of *sentiment*, and as you have demonstrated full well, the treasury is next door. We of more laborious and less remunerative callings do not envy you; we congratulate you upon the wise choice of a specialty.

But beyond all *persiflage* and in all seriousness, we are proud of the work you have done, and are doing for the sake of humanity, and of our common science. We are proud of the records of our own Academy in your fields of investigation, of Greene, Elsberg, Agnew, Noyes, Loring, Sexton, O'Dwyer, and others who have crossed over the river, and are resting beneath the shade. We are equally proud of the long list of living workers in your society whose names are already known to fame.

As President, and for my fellows of the New York Academy of Medicine, I extend to you a hearty welcome.

PRESIDENT'S ANNUAL ADDRESS.

WENDELL C. PHILLIPS, M. D.

For the thirteenth time, in annual conclave assembled, we are about to fulfill the purposes contemplated by the founders of our Society as outlined in our Constitution, as follows:

"The object of this Society shall be: the promotion of the science and art of medicine, especially in its relation to laryngology, rhinology and otology; the development of its members in original scientific work and the promotion of social intercourse."

Today, therefore, in larger numbers than ever before in our history, hailing from Maine to California, from the Provinces north of us to the Lone Star State in the South, and with hands across the sea in touch with honored co-workers of other lands, we are to join in presenting the results of our ripe experience, patient research and painstaking observation, and with friendly debate and kindly criticism to so emphasize and crystalize the truth that not only our own labors shall be set upon a higher plane, but through our published transactions the world of Laryngology, Rhinology and Otology may be aided in the monumental search for truth.

The three nations especially represented here today, friendly as they are in all their relations, and particularly so in matters pertaining to science, are far in the vanguard in achievements in the specialties which we have the honor to practice. Indeed, so friendly are there relations, both national and scientific, that with one accord, with a single bumper, and in the same breath, we may give utterance to the national salutations, "Hoch der Kaiser," "God Save the King," and, in our more youthful American enthusiasm, "Hurrah for Teddy Roosevelt!"

Among the pleasures associated with the presidential office is that of extending a cordial greeting to the members and guests who honor us by their presence upon this occasion, and on behalf of the members residing in New York and vicinity I give you the hand of welcome to our city, and also to this notable building which has been dedicated to the science of medicine and surgery.

The New York Academy of Medicine, with its active membership of one thousand, with its thirteen sections, each holding monthly meetings, supplemented by bi-monthly general meetings, and with the second largest medical library in America, opening wide its doors to all national societies free of charge, is a monument to medicine of which all New York physicians are justly proud.

My worthy predecessors in this honored office, aided by councils composed of our ablest men, have conducted our affairs with marked wisdom, safeguarding our membership, outlining our scientific programs, and exercising a wise watch-care over our transactions, with the result that in a few short years we have reached an enviable position among our national medical bodies.

It has been my pleasure to be present at all the annual meetings, and to be officially connected with the Society from its inception. This relationship, continued by your pleasure, has enabled me to closely watch our growth. The duties have been made pleasant and the burdens light as a result of the cordial support, friendly greetings and kindly encouragement of the entire membership, and whenever the years of decline shall come, as come they must, not the least among my reminiscences will be the great pleasure derived from your confidence and friendship in desiring me to remain so long in an official relationship, which has finally resulted in the bestowal of the highest honor in your power to confer. Need I say more of my appreciation?

During the present year four section meetings have been held. Your president was able to attend three of them. These meetings were all of high order, with discussion so free and full that it was difficult to complete the programs; indeed, at one meeting several papers were not read for lack of time. A total of sixty-three papers appeared upon the four programs and many of them gave evidence of marked ability and originality. From every possible standpoint the results have shown the wisdom of maintaining the section meetings. In fact, with our present membership it would be impossible with a single annual meeting to arrange a program which would permit the reading of so many papers and the opportunities of many of our members to present communications would therefore be proportionately handicapped. The section programs are necessarily largely miscellaneous in character, and they should remain so in order that individual members may bring into prominence the results of their special work along any line.

Under our By-laws the transactions of the sections may be edited and abstracted by the publication committee.

The section meetings benefit the Society in another way not usually appreciated by the membership at large, in that the council is thus left free to construct the program for the annual meetings. This allows full and comprehensive presentation of topics of widespread interest and importance to the membership, and all may be done in proper sequence. The present program is largely of this type, the main topics having been decided upon by the Council after mature deliberation. It is my belief that this plan should not only be followed but perfected; and in the future it may even be found wise for the Society to vote upon the topics which are to be presented at its annual meetings, and even to suggest the names of those who should be invited to prepare the scientific papers. With this general plan for the construction of programs I am in full accord.

It is fitting at this point to commend the work accomplished by our four vice-presidents this year. A vice-president in this Society is not elected for purposes of ornamentation, or simply to assume the duties of the president in his absence, but he must arrange for and conduct the scientific meetings of the section which he represents. The results of the labors of your vice-presidents for the present year have given full evidence of your wisdom in their selection.

At the last annual meeting the Society appointed a committee to make a much-needed revision of our Constitution and By-laws. During the year this committee has given much labor to the work of revision and their report, which is in your hands, recommending amendments, has received the full approval of the Council, and now awaits your consideration and vote. I heartily recommend the adoption of all the improvements and changes suggested by the committee.

It becomes my sad duty to chronicle the death of two of our members, Doctor S. E. Solly, of Colorado Springs, and Doctor William P. Brandegee, of New York. A third death from our former members is that of Dr. J. B. Stone, of New York, who resigned his membership about two years ago on account of continued ill health.

Dr. S. E. Solly was the fourth president of this Society. He practiced his profession in England until ill health necessitated his seeking a different climate, and finally settled in Colorado Springs, Colo., in 1874. Here he ministered to the suffering multitudes who, like himself, had journeyed to this promised land. He was kindly in spirit, gentle but yet manly in manner, with an ever-ready wit, and equipped with a good mind and proper preparation for his life work. He succeeded not only in winning the highest esteem of his patients, but in gathering to himself the warmest friendship of his professional brethren.

William P. Brandegee! How the mention of that name thrills those of us who labored by his side in the hospitals and societies, where one's real nature crops out in the lesser details of almost constant association. His deep, melodious voice always struck a joyous note. When adversity came he was heroic, cheerful, and without cowardice or faltering. I never heard one unkind word spoken of him. He served as an assistant surgeon, also first in the Manhattan Eye, Ear and Throat Hospital, and at the New York Eye and Ear Infirmary from 1896 until the time of his death. The esteem in which he was held by his co-laborers in the New York Eye and Ear Infirmary was beautifully shown by having his chair in the clinic room vacant for over thirty days subsequent to his death, during which time a large vase upon his cabinet was daily replenished with beautiful flowers. Just as he was about to reap the full fruits of his constant toil the Great Reaper gathered him into His harvest.

The Council has appointed members who have been requested to prepare obituaries to be presented at this annual meeting.

A word as to our recent achievements. A little more than one year ago the Council set apart from its funds a sum of five hundred dollars designating it as an original research fund, from which a reward could be made to any member whose labors in the field of Laryngology, Rhinology or Otology should be productive of results showing originality. This was not done for the purpose of remunerating a member for his labor, but to enhance and dignify his achievement by a substantial acknowledgment, designed to confer upon him especial honor and incidentally to partially reimburse him for his necessary outlay in conducting experiments and research. One carefully prepared essay was submitted last year, but was not deemed quite up to the standard required by the Council in conferring this prize. During the present administration no essay has been submitted.

It is a pleasure also to state that the Society was enabled to set apart a further sum of one thousand dollars as a foundation for a permanent fund. This amount has been placed at interest and it is hoped that we may be able to add to it from time to time until it shall reach an amount adequate to produce an annual income sufficient to cover at least the expenses connected with all prize essays and scientific achievements. The treasurer informs me that the balance in the treasury at the present time is sufficient to warrant the addition of a further sum of $1,000 to this permanent fund. In order that we may legally hold and invest such funds, steps have been taken to have our Society incorporated.

My predecessor, in his masterful annual address, expressed a hope that ere long our Society would take steps to publish its own journal, predicting that "such a journal would immediately take its place as a recognized authority, and would be read by every laryngologist, rhinologist and otologist from the Atlantic to the Pacific," and stated that he was firmly convinced "that it would be self-supporting from the start, and as it would be the natural channel for the Society's transactions it would save us the separate cost for our annual publication, which is no insignificant item in our yearly budget." His recommendation was taken up by the Council and a committee appointed to report at the next annual meeting. The only real objection raised thus far has been the fear that it might be a failure financially. I heartily concur with

Dr. Logan's views, and sincerely hope that in the near future we shall publish a journal, the standard of which shall be of the highest order, devoted to the specialties which we have the honor to practice.

Should it be deemed impracticable for any one society to assume the responsibilities connected with the publication of a journal, the desired end might be accomplished by enlisting three or more of the American societies in the project.

This Society, during its entire existence, has been singularly fortunate in the selection of the members who have served upon the Council, and the past year is no exception. Your President acknowledges his great indebtedness to this wise body who have made all needed sacrifices of time and expense to fulfill their obligations. Several meetings have been held, all characterized by harmonious action, slight differences of opinion always giving way to the majority vote, and all matters of importance have been subjected to the most careful consideration and discussion. The President has received full and hearty support upon all measures relating to the best interests of the Society.

Our new Secretary also has exhibited marked ability and has conscientiously fulfilled the great demands required by this important office.

Permit me, at this juncture, to offer a few comments upon the question of absolute accuracy and truthfulness in reporting the results of our scientific labor. Without wishing to sermonize, or in any manner to reflect upon the membership of this Society, I do not wish to plead that our entire membership, whenever reporting the results of work, shall observe the strictest truthfulness of statement, fearlessly reporting the unfavorable as well as the successful, without selection, and never claiming complete cures in cases that have been but partially successful. To be more explicit, should an operation upon an accessory sinus be reported as a cure when a discharge of pus still remains? Should a report covering a large number of radical operations for the cure of chronic purulent otitis media be published claiming cures without the qualification that in a considerable proportion of such cases more or less aural discharge continues; or should such a series of cases be reported without a mention of the fact that accidents, complications and even fatalities have occurred, although in no wise due to carelessness or lack of skill on the part of the operator? It is

my belief that the large majority of the membership of this Society are above criticism in this regard, and that they are possessed of the necessary moral courage to enable them to assume the highest possible standard in this matter. Honest errors in judgment must be expected and condoned. In the general run of published articles the experienced are usually able to read between the lines the evidences of wilful and dishonest neglect to fully report the unfavorable, but those less experienced are liable to be led into grave errors in following the teachings of men of this character. My plea is that the membership of this Society shall, with the finest *esprit de corps,* regardless of personal aggrandizement, in every instance exercise the greatest possible watch-care in their published articles, that such publications shall withhold no unfavorable results. A society with a reputation such as ours would become under such circumstances would be the envy of the world.

The following letter was recently received by the Secretary of our Society:

(Copy.)

New York, December 29, 1906.

Dr. T. J. Harris, Secretary,
American Laryngological, Rhinological and Otological Society.

Dear Sir:—Will you do me the favor to inform me whether your Society has ever adopted an official nomenclature for diseases, and if so would it be possible for you to send me a copy thereof?

While the Census Bureau, United States Public Health and Marine Hospital Services and some State Boards of Health have adopted the Bertillion nomenclature, there is still much variety in nomenclatures both of diseases and operations, and it would be a matter of interest to hospitals to have a nomenclature come from so an authoritative a body as yours.

Thanking you for any information you may be able to give me in the matter, I am,

Very truly yours,
(Signed) S. T. Armstrong.

General Medical Superintendent, Bellevue and Allied Hospitals of New York City.

This letter calls attention to a matter of much importance. During the past two years I have had occasion to scan many of our

text-books, journals and reprints which are published in the English language, and more recently I have compared the nomenclature employed in several of our hospital reports wherein diseases and operations are recorded. The variations in nomenclature are many, resulting in considerable confusion. It would add much to the harmony of our publications if an official nomenclature were adopted, and I recommend that a commitee of this Society be appointed to undertake this work, with the power to request representatives of similar societies to co-operate. By this means it will become possible to devise a nomenclature covering the diseases and operations of the ear, nose and throat which can be adopted and employed by English-speaking otologists, rhinologists and laryngologists. I further recommend that a reasonable sum be drawn from our treasury to cover the necessary expenses of said committee.

It was my original purpose to incorporate in this address a brief résumé of the year's progress in Laryngology, Rhinology and Otology, but the time limit had already expired, and I must give way to the fruitful scientific program prepared for you.

It is a pleasure to welcome one of our distinguished foreign members in the person of Prof. G. Killian, who has journeyed to America in response to our invitation to present in person the results of his fruitful labors in the specialties covered by this Society. Many of us have been his pupils, and all have perused his writings. We give him our heartiest welcome and hope that his visit to America will convince him of the sincerity and earnestness of his confreres in the younger country, and of their desire to place these specialties upon the highest possible plane of scientific progress.

In conclusion, allow me to once more express my keen appreciation of the honor conferred in choosing me to preside over your deliberations. Hoping for your forbearance, and that my shortcomings may not mar the tranquillity which should attend the official proceedings of this body, I now invite your attention to the scientific program which has been prepared for your consideration, and declare the thirteenth annual meeting of the American Laryngological, Rhinological and Otological Society open for business.

PAPER:

MODERN METHODS IN THE REPAIR OF CLEFT PALATE.

JOHN B. ROBERTS, A. M., M. D.

Much improvement has been made in recent years in the management of congenital clefts of the palate. Fissure of the soft palate is not a very serious deformity, and may usually be repaired by a plastic operation without special difficulty. Freshening the edges of the cleft by paring or by splitting the tissues and applying sutures tied not too tightly may be sufficient. If such operative measures are unlikely to be followed by union, because of tension of the stitches, the surgeon may make flaps of half the thickness of the soft palate and thus bridge the gap.

On this occasion, I am considering especially the treatment of clefts of the hard palate. This condition is usually accompanied by cleft of the soft palate and often by cleft of the alveolar process of the jaw as well. Single or double fissure of the upper lip, the so-called harelip, is very frequently associated with the imperfect palate.

These congenital anomalies of development have been the subject of most surgical discussion, and to a certain extent have heretofore been the despair of operators. Operative treatment has often been unsatisfactory, because of the impossibility of obtaining and maintaining an aseptic wound in the mouth and nose, and because the tissues at hand for closing the gap have been insufficient to permit osteoplastic reconstruction without tension on the sutures. Efforts to complete the partition between mouth and nose by means of obturators with an attached movable velum have not been altogether successful. Such applicances cannot be adjusted in infancy, must be increased in size as the child grows in stature, and would probably be difficult of adjustment in the adentulous mouth of the aged.

The problem before the surgeon is to close the abnormal communication between mouth and nose so effectually as to obviate the passage of food and air through it. Unless this is done, the infant

is nourished with difficulty, and its upper respiratory tract becomes affected with chronic catarrhal inflammation. Later its nasal chambers and its naso-pharynx fail to obtain normal development, its speech is exceedingly defective, and its facial outlines lack normal contour. As a secondary result of the palatal deformity, the cleft upper lip, when closed by operation, becomes an expressionless band stretched across the teeth. The lower lip is so prominent in contrast to this reconstructed upper lip that the mouth is ludicrously deformed, unless a large V-shaped section is removed from the bulky lower lip. In some cases it may be wise to increase the size and projection of the upper lip by inserting into it a portion or portions of the lower lip or cheek.

I shall now state my views as to the most satisfactory way of solving the operative problem presented by clefts of the bony palate. I use the term operative problem, because I believe that obturators and artificial substitutes for the hard and soft palate should be rejected as inferior to plastic reconstruction.

A. Brophy is correct in his assertion that in most, if not all, cases the cleft is due to a separation of the two halves of the roof of the mouth and not to an actual absence of the bony tissue. The congenital fissure is a want of union of the palate processes of the two upper maxillary and the two palate bones and the inter-maxillary bones. The partition wall between nose and mouth will usually be re-established, if the separated halves are forced into apposition. If the infant's mouth is closed so that the relation of the alveolar arch of the mandible to that of the upper jaw may be seen, the abnormal spreading of the upper alveolar halves will be quickly appreciated. It is not improbable that the "bite" of the mandibular alveolus inside of the upper alveolar arch may tend in the earliest months of infancy to force the lateral halves of the upper jaw still further apart and thus increase the cleft in the palate.

B. Acceptance of the propositions just stated compels the operator to grant that the bony roof of the mouth should be re-created as soon as possible after birth, provided this is possible without grave risk to life. A simple and most efficient method of doing this is to carry wires through the two segments of the upper jaw, force them together while they are semi-cartilaginous in structure and hold them in contact by twisting the ends of the wires over lead palates.

This operation devised by Brophy is philosophically and mechanically correct. It is surgically wise, because it is quickly performed, causes little hemorrhage, and gives rise to no shock of importance. It should be performed within a few days or weeks after birth, although in neglected cases its performance is possible, perhaps, up to six months. At that time the cartilaginous jaw usually becomes too rigid from ossific deposits to be thus drawn into proper shape. Sometimes the coaptation in these late cases may be obtained, but only after dividing the jaw above the alveolus with a chisel or saw. This increases the gravity of an otherwise safe operation, because bleeding and shock are augmented. It is not improbable that children of six or more months are more shocked by simple operative procedures than newly-born infants.

After the wire "tie-beams," as they may be called, have been left in position for a couple of months, the bony roof of the mouth will be firmly reconstructed. The wires may then be removed, and the fissure in the soft palate closed with sutures. The edges of the bony cleft may be freshened when the wires are inserted; but this step is not essential. If the margins touch, the pressure will soon cause erosion and adhesion. The soft palate may be repaired at the time the hard palate is operated upon, if the operator thinks it judicious to prolong the operation at that time. It will readily be understood that the probability of success after suturing the soft curtain of the palate will be greatly increased by reason of the previous approximation and closure of the cleft in the bony palate. The upper jaw has been made much narrower and the soft parts have been carried towards the middle line at the same time. Therefore there will be comparatively little strain on the sutures which draw the soft tissues together. The margins of the cleft may even be in contact before the edges are freshened and the stitches inserted.

C. The error of leaving cleft plates untreated by operation until the patient is two, three, or more years old should not be committed. It is important to remedy the abnormal width of the roof of the mouth while the bones are sufficiently soft and pliable to be bent into place by the surgeon. This abnormality in the relative width of the upper and lower jaws should be corrected therefore in earliest infancy. The partition between nose and mouth must be complete in early life, so that the nasal chambers, their accessory sinuses, and the naso-pharynx may be developed—that is expanded

—by giving to respired air the normal, or physiological, direction of its current. The cavities of the face connected with the function of respiration cannot be properly enlarged during the growth of the infant so long as mouth and nose admit air in breathing as one merged opening.

The existence of cleft of the hard and soft palate not only contributes to nasal, aural and pharyngeal catarrh, but may, perhaps, increase the liability to pulmonary infections. Its grave, and almost insurmountable, danger to proper speech has always been recognized; but the interference with the facial contour and the physiological needs of the voice, respiration, and dental occlusion have been appreciated only recently.

D. When early operation has been admitted and the delay has therefore allowed the displaced upper jaw bones to become ossified in their unnatural position, the wire-tie-beam method cannot be used. It then becomes necessary to cover the gap between the bones by muco-periosteal flaps obtained from the oral or inferior surface of the palate processes of the upper maxillary and palate bones.

This operative step should be done in early infantile life. It is desirable to undertake it before the molar teeth are erupted. It should be done early because the opening between mouth and nose should be closed as soon as possible in the child's life for the physiological reasons already mentioned. It is often best accomplished before the molar teeth have appeared, because one of the flaps may then be cut so large as to include the mucous membrane overlying the adentulous alveolus.

E. In this stage of the treatment I prefer to use the form of flaps suggested by Lane. He makes a crescentic antero-posterior incision on the roof of the mouth close to the alveolus. This is carried through the mucous membrane and periosteum. A muco-periosteal flap is then raised by blunt dissection from the bone, to be subsequently laid over the cleft in the palate with its raw surface towards the mouth. No incision is made along the margin of the cleft, because the flap obtains its blood supply from this region. On the other side of the cleft a linear antero-posterior cut is made, through the mucous membrane and periosteum, along the *edge* of the fissure, and a flap is raised from the bone by undermining. The edge of the everted flap from the opposite half of the palate is tucked under this undermined flap and secured by two rows of fine sutures introduced by means of very small sickle-shaped needles.

The flap, which is to cover the fissure of the palate, and which is everted, is cut from the wider portion of the bony palate. If there be no molar teeth present, it may be made very large, when necessary, by including in its area the fibro-mucous membrane covering the alveolus and even some of the mucous membrane lining the cheek beyond the alveolus. The soft palate tissues are treated in a similar though not identical manner.

It is possible that Brophy's method of performing this stage of the treatment is as satisfactory as Lane's. I have had no experience with it. He raises two muco-periosteal flaps by undermining the tissues on the inferior surface of the fissured bony palate. Each flap is raised by an incision made at the side of the cleft. There is no cut made near the alveolus as in the Langenbeck and the Fergusson operations. The undermined flaps are brought together edge to edge in the middle line. Wire sutures carried through the flaps near their attachment to the bone are passed through leaden plates, or splints, and twisted. These tension sutures and splints steady the soft flaps and lessen the tendency of the sutures, which oppose the raw edges of the flaps, to cut out.

In both of these methods the soft palate should be freely detached from the posterior edge of the hard palate before the sutures are inserted. This step is of the utmost importance. It permits the velum and uvula to drop towards the mouth and greatly facilitates the repair of the cleft without tension on the stitches.

The palatal muscles at the hamular process should not be divided by a tenotome as suggested by Ferguson. This procedure, formerly a good deal used, is not necessary in these modern methods, and may be disadvantageous to the patient.

The harelip should, as a rule, not be repaired until the palate has been reconstructed. If the palate operation is done in the early weeks of infancy, the parents will not be averse to postponing the operation on the disfiguring harelip until the condition within the mouth has been remedied.

PAPER:

OPERATIONS FOR CLEFT PALATE AND THEIR RESULTS, ESPECIALLY IN RESPECT TO THE IMPROVEMENTS OF SPEECH.

G. HUDSON-MAKUEN, M.D.

An operation for a cleft palate is something like the holy state of matrimony, in that it should be entered upon advisedly and only after prayerful consideration. It is like it also in respect that if at first we don't succeed, we try, try again. One of my patients was operated upon under general anaesthesia eight times and another seven times. I may say that in neither instance did I do the operation myself. In fact, I have never been able to persuade my patients to submit to so many anæsthesiations for the correction of a single deformity. When several operations are likely to be necessary the parents or patient should be so informed.

In the difficult cases I am inclined to think that the operation described by our colleague, Dr. James F. McKernon, may be the preferable one. A preliminary tracheotomy is done and the patient breathes through a tube during the operation and during the process of healing. The nasal and oro-pharyngeal cavities are packed and the wound is treated antiseptically as after any other operation. The objection to this procedure, of course, is the fact that it adds to the cleft palate operation a second one of some importance and gravity, but this objection may be more than offset by the completeness and permanency of the closure of the cleft when the parts are kept comparatively clean and at rest during the process of repair. I have had no personal experience with this operation, but I should be inclined to favor it in those cases in which more than one, or at most two operations by the simpler methods might be necessary.

In the technique of the cleft palate operation there is yet much to be desired, although it has had ample time for improvement. The operation for the resection of the nasal septum is younger by about 140 years, and yet its technique easily excels that of the operation

under discussion. This is probably due to the fact that the cleft palate operation goes for the most part to the general surgeon rather than to the oral and nasal surgeon, and, therefore, too little interest is taken in it, and suitable instruments and appliances have not been devised.

The anaesthesia apparatus of Dr. Fillebrown, modified and improved by Dr. Mary Rupert, is a device that should be of value in the way of improved instrumentation and if it could have a syphon pump attached for the removal of mucous and blood from the oropharynx, similar to the so-called saliva ejector employed by dentists, it would result in the saving of much time and trouble.

The operation is of sufficient importance also to require trained assistance, such as we find so necessary in other operations in the region of the respiratory and alimentary tracts. We must remember that it is not an easy operation, and that there are many little things that may happen to thwart our purposes. Before deciding upon the best procedure for any particular case it is well to consider whether any operation at all should be attempted. Those of us who have had experience in this work have doubtless operated in many instances when we have later wished we had withheld our hand and when no kind of operation, however skillfully performed, would have succeeded in bringing about satisfactory results.

There are two reasons for the desirability of closing a palatal cleft. One is for the improvement of the general health of the patient, and the other for the improvement of the voice and speech. As to the improvement of the physical condition by the closure of the palatal cleft there can be no question. Nature did not intend that the oral and nasal cavities should be without a partition wall, else she would have supplied them with similar anatomical structures. The sensitive peripheral nerves of the nasal cavities render them a most unsuitable and uncomfortable passage way for the various kinds of food, and the nerve supply of the oral cavity renders it entirely unsatisfactory for respiratory purposes.

When the palate, which forms the partition wall between these two cavities, is cleft, much of the food and drink of the individual must of necessity pass through at least a portion of the nostrils, and that the most sensitive portion, namely, the region at the junction of the middle with the posterior third, and much of the inspired air must pass through a portion of the oral cavity without being warmed, moistened and cleansed, by normal intranasal structures.

As a result of these two factors there is invariably a marked catarrhal condition of the nostrils and pharynx, and this is generally a fetid catarrh that vitiates both the air that is breathed and the food that is eaten. But deleterious as this may be, and is, upon the health and morale of the patient, the fact that he is branded for life by the peculiar character of his speech and its inefficiency as a natural means for the expression of any thoughts he may chance to have, is quite as great a factor in the prevention of normal physical and intellectual development.

The two reasons, therefore, for attempting to close a cleft palate, are first, to improve the physical condition of the patient by giving to him a more nearly normal respiratory tract, and second to improve both his physical and mental condition by giving to him an approximately normal means for communication with his fellows, through the channels of oral expression. The second reason for the operation is even more important than the first, because it effects the individual both mentally and physically.

My experience has been that the mere closure of a cleft palate in the adolescent or adult person does not as a rule improve the speech to any appreciable extent. I am aware that there are those who hold an opposite opinion, but in the cases that have been improved I think some outside assistance has always been rendered. Even the little help that may be given by an intelligent parent is fraught with good results in many instances, but the degree of success that may be obtained is generally proportionate to the skill of the teacher and the ability of the patient for persistent and concentrated effort. It has been said that a faulty habit of speech must be supplanted by a correct one, but it is more than a habit. It is a deeply seated neuro-muscular disturbance or perversion that has arisen from an effort on the part of the mechanisms of speech, central as well as peripheral, to accommodate themselves to developmental structural irregularities in certain important parts of these mechanisms. It is somewhat analogous to the effort on the part of the neuro-muscular mechanisms of the heart to accommodate themselves to a faulty valve, but it is far more complicated, because of the volitional and other psychical faculties employed in the development of speech.

The palate is one of the most important organs of voice and speech. Its integrity is essential to the tones of the voice as well as to the conversion or moulding of voice into speech by the processes of so-called articulation. The soft palate has a wide range of move-

ment. Its function in vocalization is to assist in regulating the size and shape of certain important resonance chambers, and its function in articulation is to shut off the nasal from the oral cavity during the emission of the explosive and fricative sounds, and to form contacts with the tongue in the formation of the so-called posterior linguo-palatal sounds.

PHYSIOLOGICAL ALPHABET.

CONSONANTS.

	Voiceless oral.	Voiced oral.	Voiced nasal.	
Labials.	P. Wh.	B. W.	M.	Paul Brown made white wax.
Labio-dentals.	F.	V.		Full voice.
Linguo-dentals.	Th′	Th″		Think thou
Anterior, Linguo-palatals.	S. Sh. T.	Z. Zh. D. L. R.	N.	Some zealous sheep leisurely took down nine large rails.
Posterior Linguo-palatals.	K. H.	G. Y.	Ng.	Can girls bring home yeast.

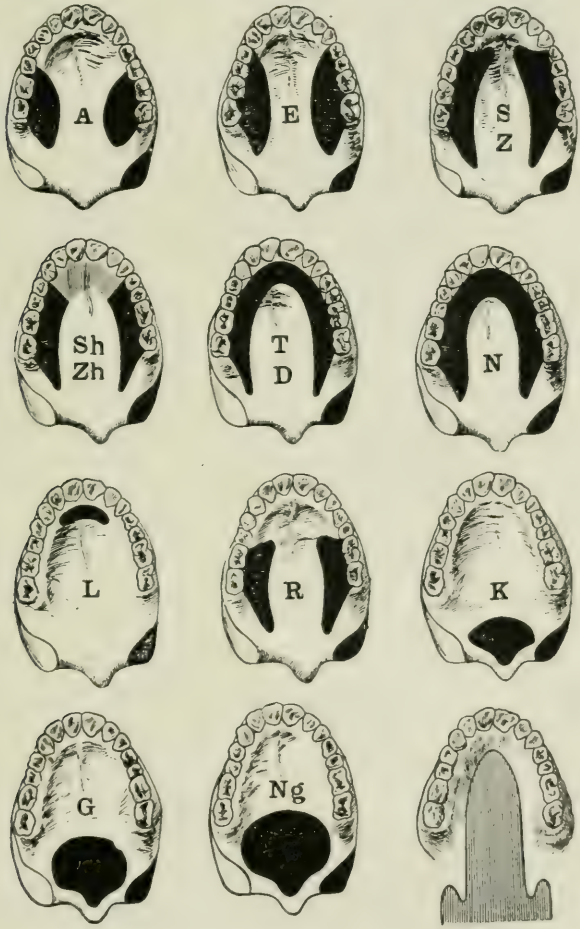
VOWELS.

ā	ā	le	long	ō	ō	ld	long	a͞r	f	a͞r	e	o͞r	f	o͞r	e
ă	ă	t	short	ŏ	ŏ	n	short	a͡r	f	a͡r		o͡r	f	o͡r	
ä	ä	lms	Italian	o͞o	o͞o	ze	long	e͞r	h	e͞r	e	o͡or	p	o͡or	
a	a	ll	broad	o͡o	l	o͡o	k	short	e͡r	h	e͡r		u͡r	p	u͡r	r
a	a	sk	intermediate	ĭ	ĭ	t	short								
				ŭ	ŭ	p	short								
ē	ē	ve	long												
ĕ	ĕ	lk	short												

COALESCENTS.

This will be better understood if we glance for a moment at the charts which I show you here. (Chart No. 1.) No. 1 contains the Physiologic Alphabet or table of vowel and consonant sounds. You will observe that the consonants are arranged in groups and named

according to the particular organs of articulation employed in their formation. In chart No. 2 we have a series of drawings of the palate, alveolar arch and teeth, and the shaded portions represent the points of contact of the tongue during the emission of the sounds, represented by the letters or symbols accompanying them. I may

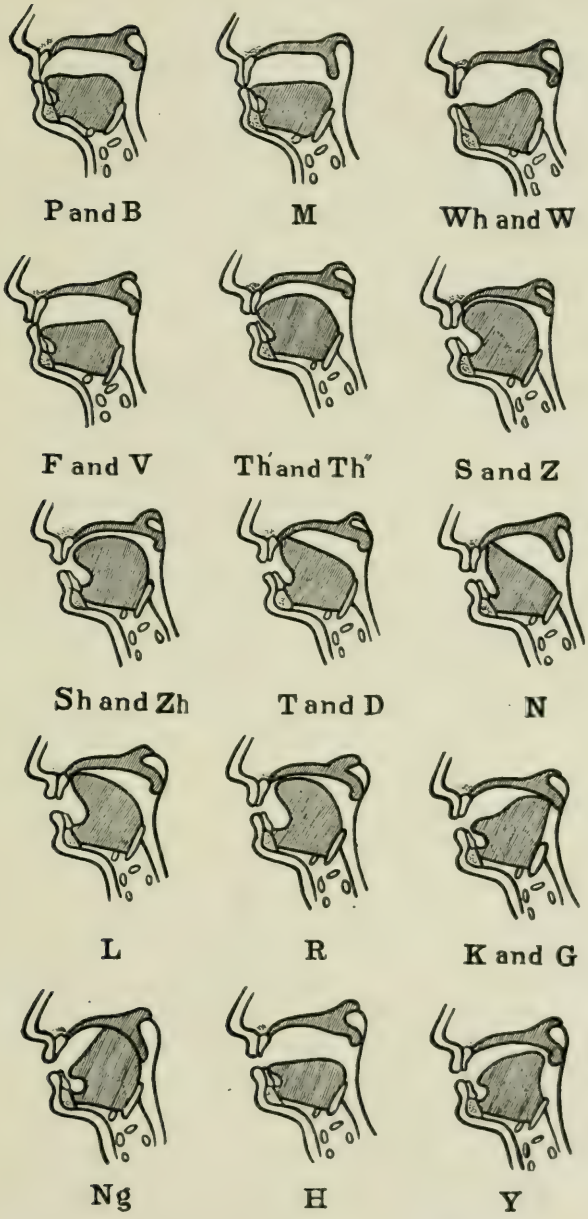


say that these drawings show fairly accurately the exact points of contact of the tongue with the palate, for they are taken from Kingsley's palatograms, and I have only slightly modified them to make them accord with some results that I have obtained from similar studies and experiments. The last drawing on the chart is one showing a complete cleft of the palate.

If we compare the normal palate with the cleft palate in the drawings and glance at the points of tongue contacts we shall readily see exactly what consonant sounds must be faulty when the palate is cleft, and these, of course, are the sounds that we hope to improve by our operation. But in addition to these there are other sounds that are defective when the palate is cleft, and they are the explosives and fricatives which require a complete shutting off of the nasal from the oral cavity. This is well shown by the drawings of some vertical sections of the organs of articulation in chart No. 3, which drawings, of course, are merely diagramatic. I would call your attention especially to the position of the soft palate shutting off the naso-pharynx during the emission of the explosives and fricatives. By a comparison of the Physiologic Alphabet with these drawings it will be seen that when the palate is cleft all the consonant sounds with two exceptions will be necessarily defective, and when the cleft extends through the alveolar arch and lip, all will be defective with no exception whatever. In the absence of the normal palate, therefore, the patient tries to substitute for purposes of speech certain other organs lower down in the throat, such as the epiglottis, the ary-epiglottic folds and the ventricular bands; and in this process of substitution faulty musculatures are developed, including a faulty development of the nerve centers supplying them. This gives rise to the neuro-muscular perversion to which reference has already been made and which, as I have said, is really more than a habit.

It must be remembered that we are dealing here with a psychical as well as a physical perversion, with a faulty development of the central as well as the peripheral mechanisms of speech, including the receptive, the executive, and even the intellectual centers.

The correction of these conditions is by no means a simple procedure. The patient himself cannot accomplish it, because his central mechanisms are involved, and he cannot diagnosticate his own case. So accustomed has he grown to his speech that his ear has come to approve it and he cannot discriminate between his faulty forms of speech and your correct ones. He has no ear for correct speech, just as some people have no ear for musical tones and have to learn them by a long and plodding process. Moreover, every day of faulty speech tends to increase these unfortunate psycho-physical conditions and to lead the patient further and further away from normal speech. Hence it is that the fewer the days of faulty speech



the better it is for the patient, and hence the importance of doing the operation, if possible, even before the developmental speech period or within the first year. The only objection to an early oper-

ation is the fact that the young patient may be somewhat lacking in vitality, and it is well known that many children in whom the halves of the palate have not united are of a low physical grade.

In the adolescent or adult cleft palate case, training will do more for the improvement of speech than will the operation itself. In other words, given a patient that can have the advantage of but one of the two procedures, I think I can give him better speech with training alone than you can give him with an operation alone. The reason for this is apparent when we consider the limitations of the operation. In the first place the speech is defective in two important particulars, namely, in resonance and in articulation. The extent to which we can improve the resonance of the voice by the mere closure of the cleft is very slight, because, however successful may be the operation, the patient will have but limited control of a short tense velum and he will be unable, therefore, to regulate the size of the opening between the oro-pharynx and the naso-pharynx. It is upon the regulation of the size of this opening, which is constantly changing during speech production, that normal resonance depends. When the opening is large, the nasal resonance predominates and when it is small the nasal resonance is diminished.

As to the other particular in which the speech of the cleft palate case is defective, namely, so-called articulation, our operation is of greater service because, as we have seen, the hard palate and velum are both essential to the normal tongue contacts of certain of the consonant sounds, and, of course, if the cleft extends through the alveolar arch and lip, nearly all the tongue contacts in the articulation of consonants will be faulty.

Not only are the tongue contacts important, but in the production of many of the consonants there is a damming up, so to speak, of the breath in the mouth, and a slight explosive effort as the sound is emitted. When this takes place in the normal mouth the velum rises and shuts off completely the oral from the nasal cavities, but in the cleft palate patient the velum cannot do this, otherwise we should be able by means of the combined operation and training to give the patient absolutely normal articulation. We can only approximate this result by uniting the velum in such a manner that it will be as large and loose as possible, with its muscles in their normal positions and relations, and then by giving the patient such exercises as will have a tendency to develop in these muscles their normal physiologic functions.

Much depends, therefore, upon the way in which the operation is done. Only the other day I saw a case in which the palato-glossus muscle of one side was united to the palato-pharyngeus muscle of the other side, and it was stretched across the middle line of the pharynx like a tight band, interfering with the normal vibrations of the voice and serving as a hindrance to the development of speech. When the muscles of an adult case of cleft palate have been united in their normal positions and relations our work has only begun, because these muscles have become atrophied from disuse; they have no so-called tonus and scarcely any power. In other words, they have lost their normal function and if left to themselves they would never regain it; and, by the way, it is this masterful inactivity of the palatal muscles after the operation that, more than anything else, gives to the speech of the cleft palate case its characteristic quality, and it is the restoration of the function of these muscles that does more than anything else to remove this disagreeable quality.

A good exercise after the operation is a vigorous gargling of the throat several times a day; and I am accustomed to try to teach my patients to acquire a voluntary control of the muscles of the palate, so that they may learn to contract and relax them at will. These exercises, together with the actual use of the palate in selected sounds of speech, will produce marvelous results, if followed up in a regular and persistent manner. I have had patients carry the work of speech improvement so far as to almost conceal the fact of any original palatal defect.

CONCLUSIONS.

1. An operation for the closure of a cleft palate should be done only when there is a fair likelihood of success.

2. It should be done only by those having acquired special skill in naso-pharyngeal and oral surgery.

3. When it is probable that several operations may be necessary the parents or patient should be so informed.

4. In the difficult cases the operation after a preliminary tracheotomy, as described by Dr. McKernon, may be preferable.

5. There are two reasons for attempting to close a cleft palate, namely, to improve the general health of the patient and to increase the efficiency of the faculty of speech.

6. The general health of the patient is benefitted in two ways: namely, by improving the hygiene of the naso-pharynx and the oral cavity, and by improving the general morale of the patient.

7. The speech is improved by a course of psychological training, in which the patient is taught first to recognize normal speech, and then to make the best use of his still imperfect organs in its production.

DISCUSSION.

Dr. James F. McKernon, of New York City, thought cleft palate work in adults could be more readily done by packing off so as to prevent the passage below of blood and other fluids. To this end, he formerly did a preliminary tracheotomy, then proceeded with the cleft palate operation. He had employed this method for the past seven years in twenty-four cases, without a death and with sixteen primary unions. By then giving the anaesthetic through the tracheotomy tube the operator was enabled to work much more rapidly. He advocated early operation and commended the excellent work accomplished by Dr. Brophy. If the patient had reached two and a half or three years of age before operation it would be necessary to teach them if any improvement in speech was obtained. He had never seen much improvement in adults following this operation, unless they were trained as advised by Dr. Makuen. He was interested in Dr. Roberts' technique, particularly as to the everted flap. He had always used the muco-periosteal flap, and in all but two cases had employed wire suture, with which he had had very good results. For the past four years instead of doing a preliminary tracheotomy, he had had the anaesthesia given by the Junker method.

Dr. Truman W. Brophy, of Chicago, believed that closure of a cleft palate by surgical means was merely following up and completing work which Nature had failed to do. In infancy there was always enough tissue to form a perfect palate, but in some way, probably in embryo, the bones had been driven apart. He regarded the preparation of the patient as essential. He had never done tracheotomy, as suggested by Dr. McKernon. The nose and mouth should be prepared by removal of adenoids and hypertrophied tonsils, and otherwise getting the field of operation in as nearly perfect condition as possible. The matter of hemorrhage was an important consideration with reference to early operation, inasmuch as there is no hemorrhage in infants. The great mortality referred to by Dr. Makuen was due largely to hemorrhage. He had not had a mortality of more than five per cent. The children who had come to him had one or all of the defects which result from the vices of parents. No sick child should be operated upon, but if the child be healthy the chances were that it would go through the operation and recover, provided the operation be done early enough. A new-born infant had bones with so little calcium salts that with the thumb and finger the parts might be pressed together, so that all that was necessary was to fix them until union has taken place. The risk was greater after the fifth

or sixth month. He emphasized what Dr. Roberts had said to the effect that a young child is not so capable of receiving shock as a child a little older. It had been the practice for years to close the lip and leave the palate open. The tuberosities of the bone and the distal molar teeth were always spread out, and the curtain of the palate was like a drum-head, so to speak; if the operation were made when the bones could be brought together, the palate would then develop normally, as it had in the patient just presented to the society. He, therefore, considered it unfortunate that the lip is closed first. The contraction of the orbicularis muscle would bring the parts in contact but would not close the fissure at the distal part. He had found that if the mouth is properly prepared before operation and kept clean afterward whatever infection might be manifest would come from the nasal surfaces, hence it had been his custom to use a spray or argyrol in the nose five or six times a day in order to keep it in as nearly an antiseptic state as possible.

Dr. Christian R. Holmes, of Cincinnati, reported some satisfactory results and some which were unsatisfactory, the latter being due to an improper selection of cases. In one case where there was some infection he had made the mistake of using hydrogen peroxid, which led to stitch infection. While Dr. McKernon had been remarkably sucessful in cases where he had made a preliminary tracheotomy, he would not himself be willing to add this additional danger to that of the cleft palate operation.

Dr. Joseph H. Abraham, of New York City, presented a patient, five years of age, upon whom Dr. Brophy had operated three years before, with perfect result.

Dr. Lefferts A. McClelland, of Brooklyn, believed the mortality of fifty per cent mentioned by Dr. Makuen very high. With the operative field in view, made possible by means of the Brophy mouth gag, which not only holds the mouth wide open but covers the tongue, pushing it out of the way, besides aiding the illumination, it was possible to perform the operation with practically no hemorrhage and little trouble from the mouth secretions, which are easily wiped away by means of sponges on sticks or by the use of the long sponge forceps of the Brophy type. Of course deftness in handling the various instruments was a desideratum of the utmost importance, and it was here that such disparity in results was noticeable. In Professor Brophy's operations the glide from one step to another was so rythmical and accurate that it would seem as if it were possible for general operators to procure his ideal results with low mortality. Such results, however, had not as yet become general.

Dr. Rorerts, in closing the discussion, agreed with those who had said that the mortality in infants under the old method might be high; with the Brophy method it was not high. He had become so discouraged with the old methods that he had at one time used

mechanical appliances made by dental experts, instead of operating; but now he felt it better to operate very early in accordance with the views and methods of Brophy and Lane.

Dr. Makuen, in closing the discussion, said the mortality mentioned in his paper was given merely to show that in the opinion of one observer the early operation was a serious matter. Personally, he did not see how the worst kind of bungling could bring about a mortality of fifty per cent. He saw no reason why the speech should not develop in a normal manner if the defect in the palate is entirely corrected before the developmental speech period begins.

PAPER:

THE PREMAXILLARY WINGS AND DEVIATIONS OF THE SEPTUM.

HARRIS PEYTON MOSHER, M. D.

There are two great causes of deviations of the septum, trauma and asymmetry of growth. Of late trauma has been held to play but a minor part. The tendency has been to make unequal development of the halves of the head the chief cause. Undoubtedly this is true in certain cases but more often the inequality of growth is confined to the bones which make the hard palate, namely, the superior maxillae, the palate bones and the premaxillae. I wish to show that irregular and delayed eruption of the teeth, especially of the incisor teeth, is the cause of this asymmetry in a large number of cases. Further, I wish to reinstate trauma as a prominent cause of deviations of the septum.

My attention was called to this subject by a paragraph in Sieur and Jacob's Anatomy of the Nasal Fossae. In the chapter on the septum, in the summary of the causes of deviations, the following statements are found; (page 42); "Hypertrophy of one of the elements making the premaxillary group of bones (the subvomer bone and the nasal spine) appears to play a great part in the formation of crests and spurs which are placed at the entrance to the nasal fossae just behind the nasal spine and appear to be incorporated with the floor. * * * Potiquet has shown that for the subvomer bone in particular the hypertrophy is connected

with the eruption of the incisor teeth which are lodged in the premaxillary bones. It seems that this group of bones, originally independent of the superior maxillary bones, undergo to a less extent than these the retrogade changes which all anthropologists have pointed out in the superior races. There can be therefore, not only a failure of parallel growth between the skull and the face but also between the different elements of the face. From this it follows that the projections and ridges of the septum anteriorly which are due to hypertrophy of the subvomer bone or the nasal spine are caused in the last analysis by the same underlying pathological cause as ridges or spurs which are placed posteriorly, namely—they are due to some disturbance in the development of the face."

I am convinced that this is true. Hypertrophy or displacement of the subvomer bone, however. can do more than cause ridges and spurs just back of the nasal spine. If there is marked delay in the eruption of the incisor teeth not only are the premaxillary wings (I prefer to call the subvomer bones by this name) thrown out of line but the cartilage of the septum is thrown out of its bed in the vomer for a very considerable distance and the septum is deviated in consequence. The starting point of a great many deviations is anterior. The force which causes them is applied anteriorly but is felt not alone anteriorly but posteriorly. At the present time I am not prepared to say just how many deviations of the septum are caused in this way, but since I have been looking into the question I have been surprised to find that so many could be assigned to this cause and I feel that continued clinical work will make the percentage of deviations so caused a high one.

Dentists for a long time have been aware of the connection between crowded teeth and deformities of the septum but as far as I have seen, in a very superficial examination of the literature, the exact relationship between the teeth and the septum has not been brought out. My work has been clinical and anatomical, an endeavor to satisfy myself as to some of the details of this relationship with but little reference to the literature except for the basal idea of the investigation, which as I said is found in Sieur and Jacob and is due to Potiquet.

In discussing my subject, the enlargement and displacement of the premaxillary wings as a cause of spurs and deviations of

the septum, I shall do so under the following headings; First, a review of the development and the anatomy of the septum; second, the changes which occur in the premaxillae as the incisor teeth erupt; third, the condition of the septum in forty cases of delayed and irregular dentition; fourth, a description of the examples of deviation of the septum found in the dissection room, where the deviations were due to hypertrophy and displacement of the premaxillary wings; fifth, asymmetry of one-half of the palate as a cause of devations; and finally, trauma as a cause of deviations.

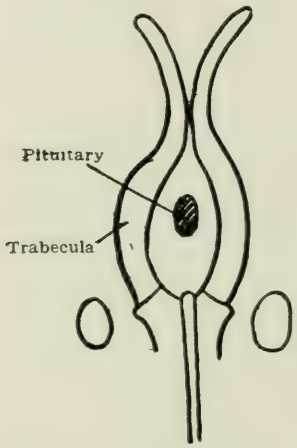


Fig. 1.

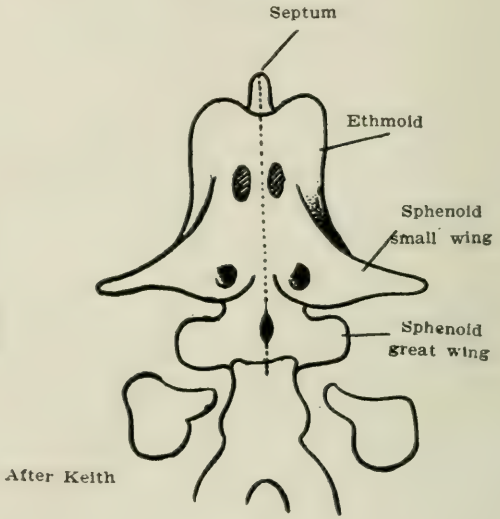


Fig. 2.

PLATE 1.

Figure 1. Diagram of the trabeculae cranii.

Figure 2. The trabeculae have widened and developed into the sphenoid and the ethmoid and septum.

The Nasal Processes. The nasal process at a very early stage in the embryo is seen to be divided into two lateral processes and two mesial processes, the latter having globular enlargements as tips. These processes are vertical septa springing from the base of the primitive capsule of the fore-brain and the parts seen on the face are the anterior ends of these septa. (Plates 1 and 2.) The mesial nasal processes fuse together and form the whole septum of the nose, the premaxillae and the middle third of the upper lip. They form also the mesial limbs of the alar cartilages.

The Vomer. The vomer is developed from the perichondrium which covers the primitive septal cartilage. A center of ossification appears in the third month at each side of the cartilages posteriorly. These fuse together below. Thus the vomer is at first a trough into which the cartilage of the septum is implanted.

The Vertical Plate of the Ethmoid. The vertical plate of the ethmoid is formed by a direct ossification of the primitive cartilage

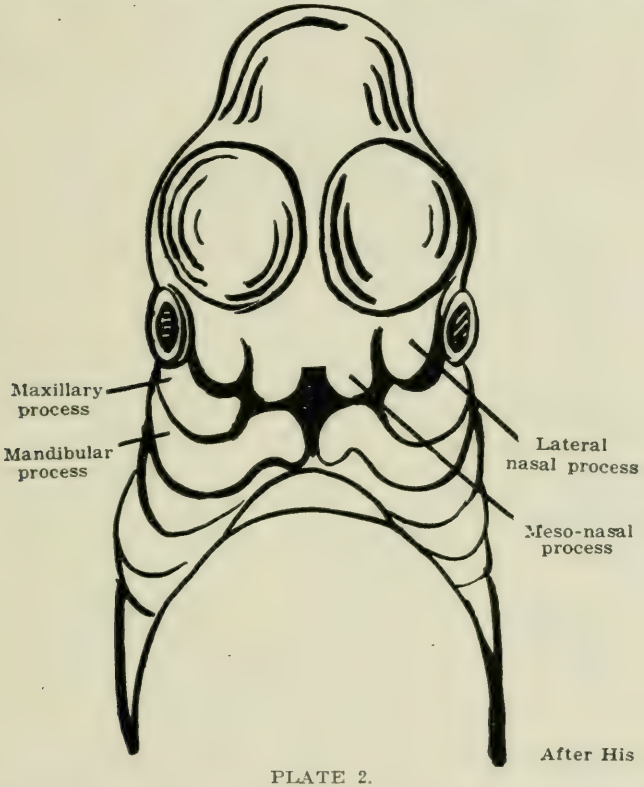


PLATE 2.

Embryo of fourth week showing formation of the face by the nasal, maxillary and mandibular processes.

of the septum. Ossification begins in the fourth month of foetal life. The crista galli, the intra-cranial part of the septum, is formed in part by ossification proceeding from the attachment of the falx cerebri.

The Premaxillae. The two premaxillary bones form the sockets for the upper incisor teeth. In the human foetus at birth the suture between the maxilla and the premaxilla can be seen on the hard

palate. (Plate 3.) It runs diagonally forward and outward from the anterior palatine canal to the alveolus between the lateral incisor and the canine tooth. On the facial aspect the premaxillae fuse with the superior maxillae in the third month of foetal life, the maxillae overlapping and almost completely excluding them from the face. In mammals generally the premaxillae are highly developed and form the snout. In the higher primates the face becomes less elongated and the premaxillae less developed. In the orang, for

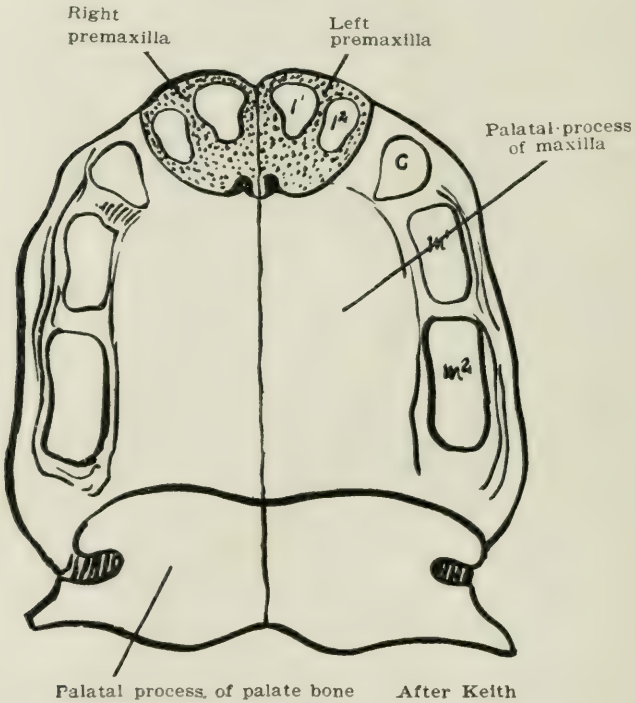


PLATE 3.

The hard palate at birth. The phemaxillary part is formed from the mesial nasal processes. The remainder by the palatial processes of the superior maxilla and of the palate bone

instance, the premaxillae are distinctly seen on the face at birth but as the permanent canines begin to be developed they fuse with the maxillae.

In man each premaxillae is usually ossified from two centers placed side by side. The two premaxillae unite in the first year after birth. Their vestigal character in man is due to the small

size of his masticatory apparatus and the consequent retrogression in the development of the facial part of his skull. (Keith, pp. 3-5.)

The result of all this is that the septum becomes ossified from behind forward. Not the whole of it, however, for the anterior part remains as cartilage. This is known as the quadrangular cartilage. I wish to lay especial stress upon two points in the development of the septum. The paired origin of all parts of the septum; and the fact that developmentally the premaxillae are as much parts of the septum as the vomer.

The Septum at Birth. The septum at birth is almost all cartilage. (Plate 4.) The only bony parts are the vomer and the two premaxillae and their processes. The vomer has a very characteristic form. It consists of two leaves of thin bone united below and

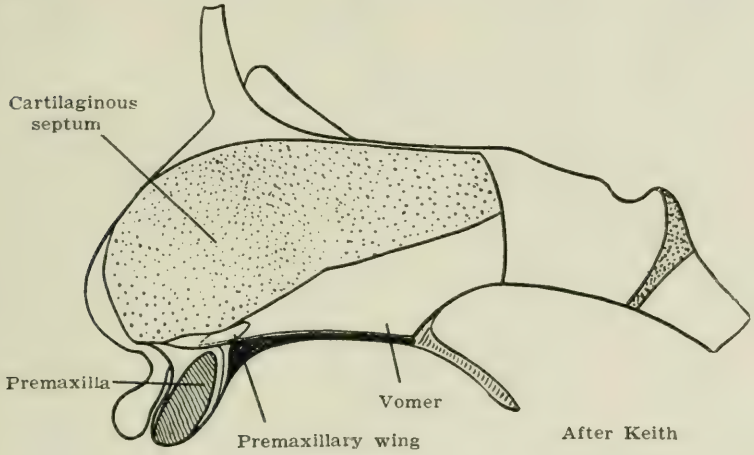


PLATE 4.
The septum at birth.

open and flaring above. (Plate 5.) This formation is a relic of its double origin, evidences of which the vomer never entirely loses. The premaxillary wings make the vomer over again in miniature. Therefore, they also form a V, or gutter. The premaxillary wings spring from the posterior half of the upper surface of the premaxillae. In the groove which they form rests the tip of the vomer. Two other processes spring from the superior surface of the premaxillae, one from each, namely the nasal spines. These again make a slight gutter, into which in its turn fits the tip of the premaxillary wings. The tip of the vomer rests in the gutter of the premaxillary wings, and the tip of the premaxillary wings rests

in the gutter of nasal spines, like the sections of the old fashioned V-shaped wooden drain. (Plate 6.)

The Adult Septum. The upper border of the adult vomer is gutter shaped like the vomer at birth, the gutter not being so

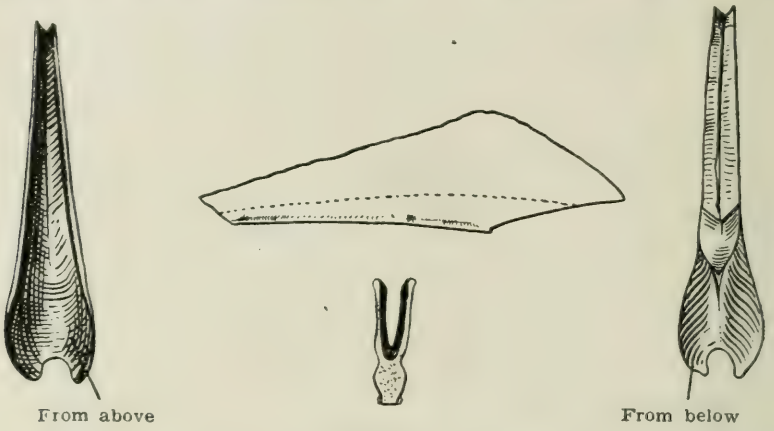


PLATE 5.

The vomer at birth, showing the two leaves and the gutter made by them.

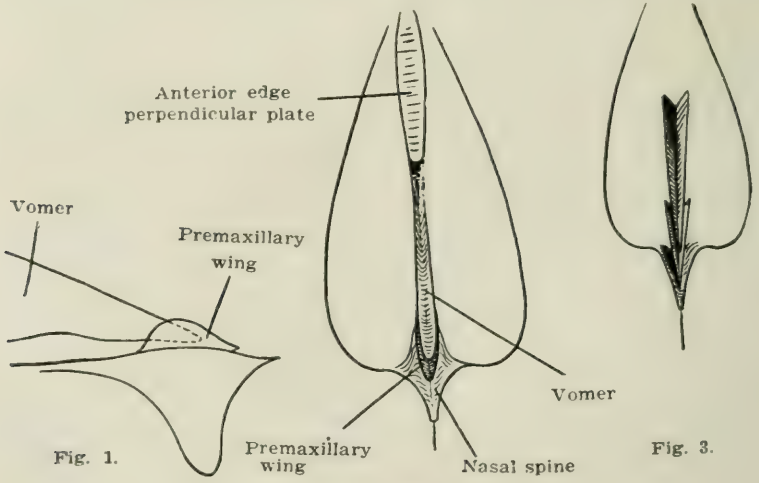


Fig. 2.

PLATE 6.

Fig. 3. is a diagramatic drawing showing how the vomer lies in the premaxillary wings, and the premaxillary wings lie in the gutter of the nasal spine.

deep. (Plate 7.) It is surprising how long the vomer retains this characteristic to a very marked degree. At four years of age the vomer still consists of two leaves for half its width. In the adult

septum the lower border of the perpendicular plate of the ethmoid is gutter shaped the same as the upper border of the vomer, with which it articulates. Between these two bones, therefore, there is a lozenge like space filled with cartilage. The strip of cartilage which fills this space is an offshoot from the posterior inferior angle of the quadrangular cartilage. It runs backward and upward in the space thus left for it, at times reaching the front wall of the sphenoid. This prolongation is called the caudal prolongation. The following are the other peculiarities of the adult septum which have a bearing

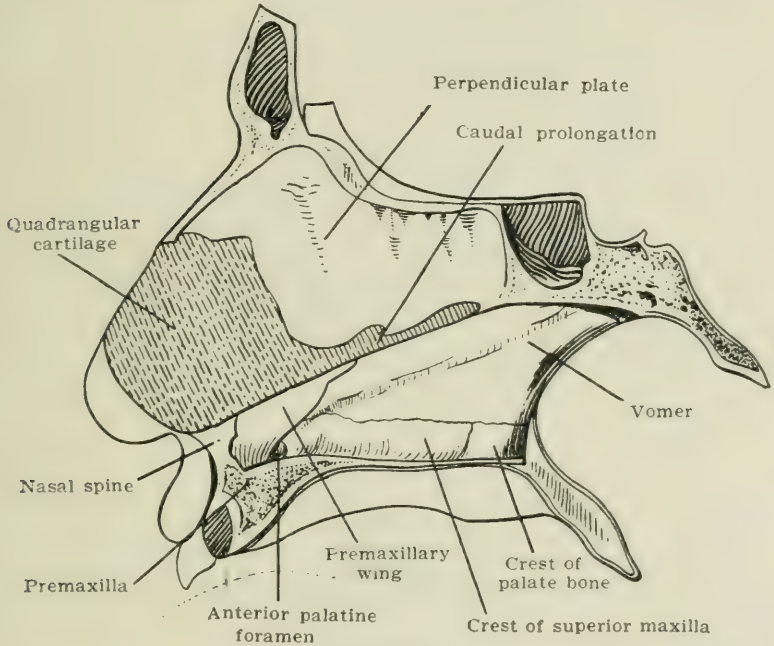


PLATE 7.
The adult septum.

upon the subject of this paper. The lower part of the quadrangular cartilage just above the premaxillary wings is very thin, whereas the upper part of this same cartilage in line with the middle turbinate is thickened into what is called the tubercle. After eight years of age the further growth of the quadrangular cartilage occurs chiefly at a center of growth just above the premaxillary wings. There is a second less important center of growth half way up its posterior border. At birth neither the palate bones nor the superior maxillae rise into a crest for the support of the lower edge of the septum. (See Plate 4.) In the adult on the other hand both

these bones have marked crests. These grow upwards while the rest of the septum is growing downward affording at times, another factor in disturbing the equilibrium of the mosaic of the septum. This factor is not generally considered.

It has usually been held that the weakest part of the septum is the articulation between the upper border of the vomer and the lower edge of the perpendicular plate of the ethmoid. From the

Fig. 1. Side view.

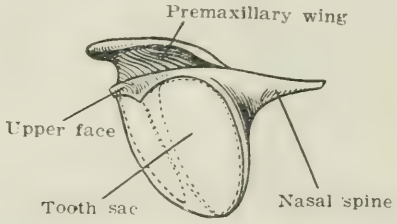


Fig. 2. Front view.

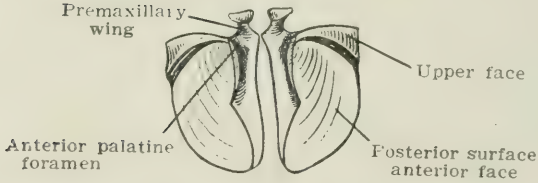


Fig 3. Premaxilla seen from above.

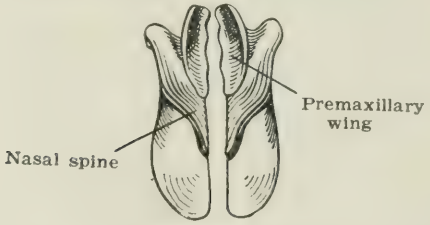


PLATE 8.

The premaxilla at birth.

description of the septum which has just been given it is easy to see why this is so. There is, however, another part of the septum which is as weak or weaker than this suture line. This place is at the tip of the vomer, or the region of the premaxillary wings. Here the mosaic of the septum is the smallest, here the tip of the vomer lies in the groove of the premaxillary wings, here the caudal prolongation of the quadrangular cartilage starts, here the quad-

rangular cartilage is the thinnest, here is placed the remnant of the organ of Jacobson, and here the quadrangular cartilage has its chief center of growth. Anatomically, therefore, this part of the septum is especially fitted to be the starting point of spurs and deviations. If for any reason the equilibrium of the septum is upset one would expect it to show itself first at this point.

The two premaxillae do not fuse until well into the first year. When they are disarticulated each premaxilla is seen to be an oval shell of bone, all the faces of which are very thin. The body of the bone is completely filled with the crowns of the incisor teeth and accurately takes its form from them. (Plate 8.) Projecting

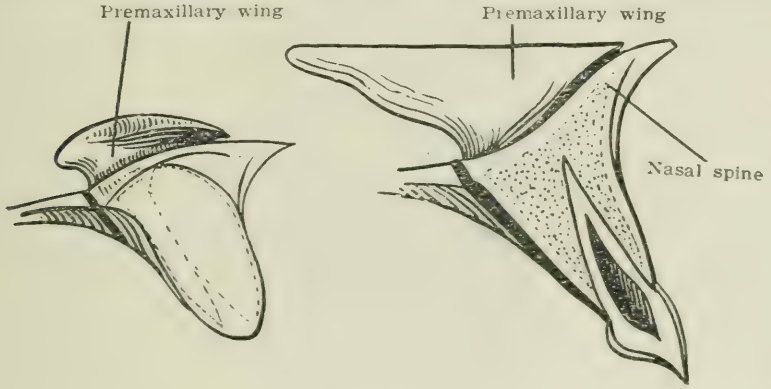


PLATE 9.

Fig. 1. Premaxilla at birth. Fig. 2. The premaxilla in the adult.

horizontally forward from the anterior edge of the upper face of the premaxilla is a pointed process called the nasal spine. The superior surface of this is slightly concave. Also projecting from the upper face of the premaxilla in the middle line, but projecting obliquely upward and outward, is another process, the premaxillary wing. This process extends along the posterior two-thirds of the superior face of the premaxilla. Its upper surface is markedly concave. The inner edge of the base of this process is irregularly undermined so that it can mortice into the corresponding process of the other premaxilla. The upper edge of the wing slopes outward and the root of the wing on its outer side is constricted by a smooth gutter, as if for a vessel or a nerve. Owing to this the wing has a narrow constricted base and a concave flaring superior surface. When the two premaxillary wings are placed together the wings mortice into one another so that they form a sizable gutter. Into

this, in the complete septum, fits the tip of the vomer also a gutter. The premaxillary wings act as cleats on either side of the tip of the vomer to hold it in place. Their position controls the position of the vomer.

From birth up to six years of age the premaxillary wings increase in size but little. From this time on, however, they enlarge rapidly, so that in the adult each wing measures about half

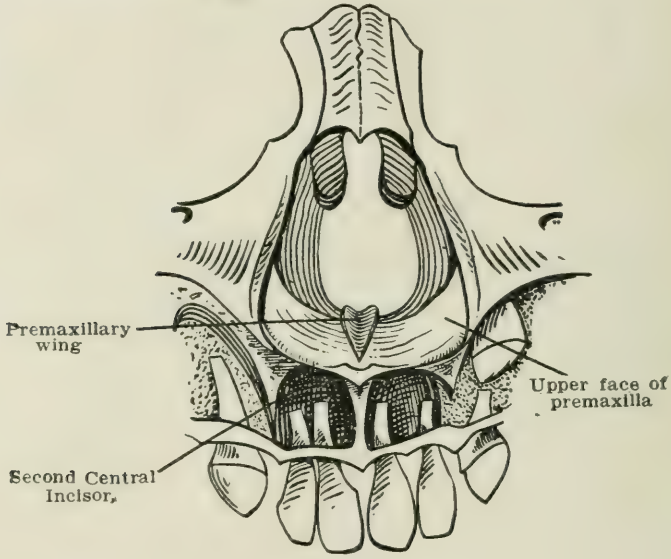


Fig. 1.

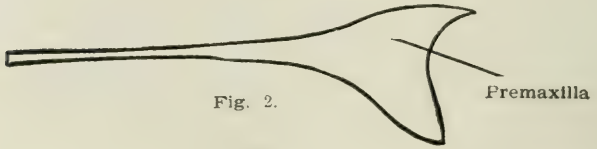


Fig. 2.

PLATE 10.

Fig. 1. Dissected alveolus of a child three years old. The upper face of the premaxilla is flat and fits accurately over the tops of the second incisor.
Fig. 2. Shows that the upper face of the premaxilla is almost flat.

an inch along its upper border and is a quarter of an inch in height. (Plate 9.) The premaxillary wing grows especially upward and backward so that its final form is triangular. The apex of the triangular piece of bone which constitutes the premaxillary wing points downward and rests just in front of the anterior palatine canal. These processes undergo a greater proportional growth than

almost any other part of the septum. This is necessary in order that the premaxillary wings should not lose control of the vomer.

The premaxillary wings are said to fuse with the tip of the vomer at fifteen years of age. On account of their position and on acount of this fusion with the vomer the two processes have usually been known as the sub vomer bones. Dissection has led me to consider them not as separate bones but as processes of the premaxillae the same as the nasal spines. At birth the premaxillary wing is firmly united to the superior face of the premaxilla by a slender neck and apparently is a part of it. For this reason I call these processes the premaxillary wings.

As the antra enlarge and descend with the eruption of the second teeth the crest of the palate bone and the crest of the superior

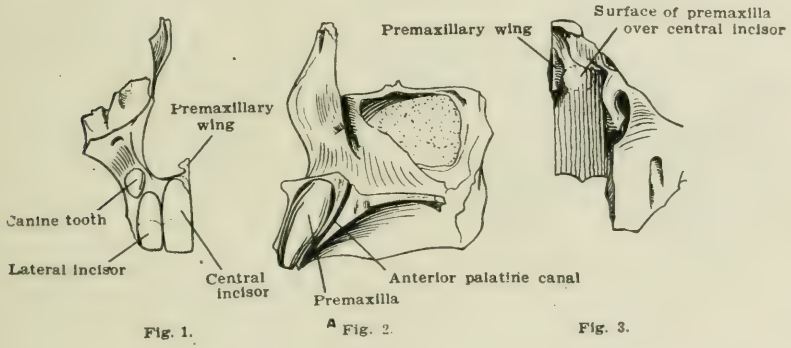


PLATE 11.

The premaxilla and the superior maxilla from a child of six.

maxilla grow upward. (Plate 10.) At the same time the nasal spines and the premaxillary wings also grow upward. Heretofore the superior face of the premaxilla has been flat like a plateau. This plateau disappears and the superior surface slopes downward and backward at an angle of forty-five degrees. The upward growth of the nasal spine is in great measure responsible for this. The spine in the adult makes a bony rim for the vestibule and turns the incisor plateau of infancy into a moat.

The fact that the inclination of the superior face of the premaxilla depends upon the teeth below it and changes as these erupt is shown prettily by dissections. At birth the upper rim of the hollow crown of the middle incisor fits snugly under the thin upper face of the premaxilla. The upper rim of the crown is flat

and so is the superior face of the premaxilla which is paper like in thickness and roofs it over. Plate 10, which is drawn from the dissected alveolus of a three year old child shows this. As the flat rim of the upper part of the hollow crown gives place to the pointed root the superior face of the premaxilla changes from a flat plateau to a slanting surface. The form of the nasal notch changes in the meantime from a triangular aperture to a heart-shaped opening. Normally, therefore, the line of the superior

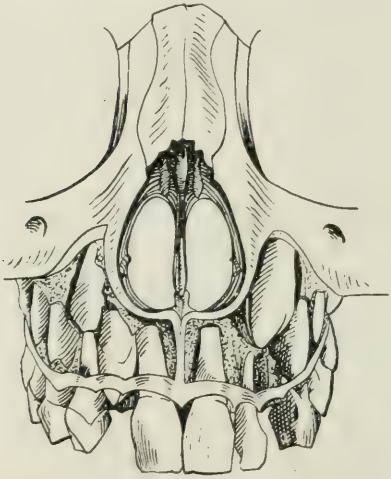
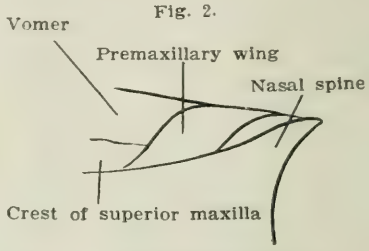


Fig. 1.



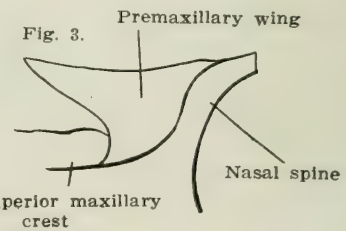


PLATE 12.

Fig. 1. Dissected alveolus of a child eight years. The nasal notch has lost its former triangular shape.

Fig. 2. The upper face of the premaxilla. The nasal spine and the premaxillary wing are beginning to grow upward.

Fig. 3. The nasal spine and the premaxilla have their adult growth.

face of the premaxilla and the shape of the nasal notch are determined by the degree of eruption of the incisor teeth. Plate 11 represents the superior maxilla and the corresponding premaxilla at six years. In this specimen the upper rim of the crown of the central incisor is flush with the under surface of the superior face of the premaxilla and occupies the greater part of it. The layer of bone over the tooth crown is still paper thin, whereas the amount of bone over the lateral incisor is very thick. The lateral incisor could have but little influence as compared with the central incisor upon the form of the superior face of the premax-

illa and upon the premaxillary wing. (Plate 12.) All other parts of the alveolus except the part made by the premaxillae are buttressed by the walls of the antrum. (Plate 13.) The superior face of the premaxilla is free in the nose and not so buttressed. The form of this, therefore, can be readily altered.

Of late the old theory that the eruption of the teeth is caused by the formation of the root and pressure upon this as it elongates

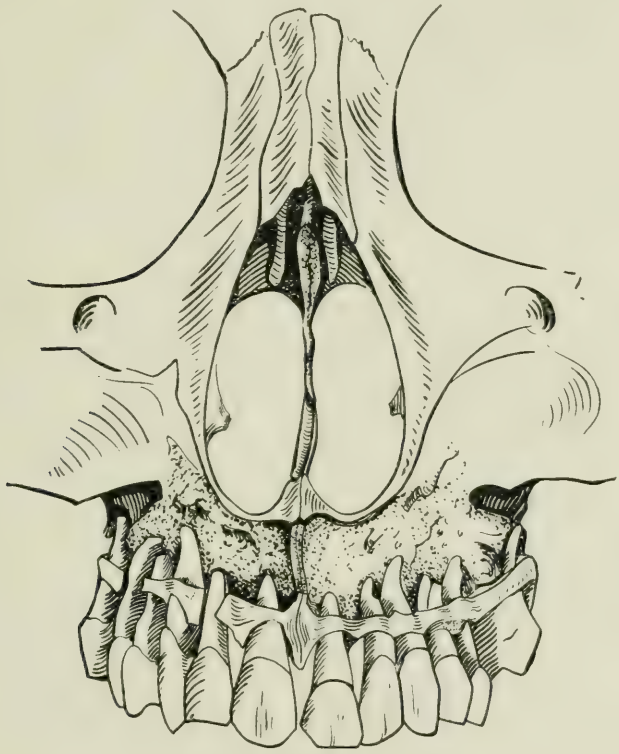


PLATE 13.

Dissected alveolus in adult. The nasal notches have the adult heart-shaped form. (See PLATE 12, Fig. 3.)

has been somewhat questioned. The new theory is that the eruption of a tooth is due to the increase of the blood pressure within the tooth sac. The tooth is forced out, so to speak, by hydraulic pressure. This may be true in part but it is not the whole truth. The anatomical facts which have just been given and the clinical findings which are to be given next in order show that the pres-

ence, or better the pressure, caused by the tooth root determines the form of the superior face of the premaxilla.

Th blood pressure theory is of interest for this reason. Hutchinson's teeth is the name of a familiar deformity of the incisor teeth, especially of the central incisors. Inherited syphilis, which causes the deformity, involves in the nose chiefly the septum. Developmentally and practically, therefore, the premaxillae are a part of the septum. The incisor teeth are likewise in a way a part of the septum; and, in common with it, suffer from the syphilitic invasion. Thus it comes about that they are selected over all the other teeth for excessive malformation. The fundamental pathological change in syphilis is an endarteritis. If blood pressure has a part in causing the eruption of the teeth the pressure would be interfered with by the arterial changes. To interfere with the permeability of an artery is to interfere with the nutrition of the part which it supplies. Therefore we generally find that all the teeth in cases of inherited syphilis are stunted.

If the normal eruption of the incisor teeth causes a change in the upper face of the premaxilla the question is at once suggested: Does delayed or irregular eruption of the incisor teeth cause a still further change, a change which is of any significance in the pathology of the septum? It does. Within the last month I have collected forty cases of irregular and delayed eruption of the incisor teeth. The patients were mostly children who came to the clinic to have their tonsils and adenoids removed. The ages of the majority were between seven years and twelve. Five of the cases were adults. Through the kindness of Dr. L. B. LeGro, of Haverhill, I had the opportunity to examine four cases from his private practice where the history of the teeth was known from the beginning and where the teeth had been carefully followed and cared for. Since every point in connection with these cases could be followed I will give the cases in detail.

T. B., female, 8 years. Eruption of the incisors six months ahead of time. Right middle incisor fully down and in good line. Left middle incisor just appearing through the gum. Both first lateral incisors lost, neither second lateral incisor showing.

Nasal examination. The arch of the palate is moderately high Both halves of the arch of the palate are of equal height. The wing of the premaxilla on the side of the left central incisor, the delayed incisor, is enlarged. There is a left vomer spur running

back from this for one-half inch. The right premaxillary wing is not enlarged. The septum deviates moderately to the right, for three quarters of an inch back.

There is no nasal obstruction. Tonsils and adenoid were removed three years ago. No tonsils are to be seen at the examination.

G., male, 8½ *years.* Both central incisors fully erupted and in perfect line, at the present time, but the patient's history card states that the left middle incisor came down six months after the right middle incisor and six months after the normal time of eruption. The arch of the palate is normal and both halves are of equal height.

Nasal examination. There is a slight vomer spur on the left, and a slight deviation of the septum to the right beginning about a third the way up on the septum and running one-half inch back. There is a slight vomer spur on the right. No nasal obstruction. Tonsils and adenoid were removed three years ago. Practically no tonsils are to be seen.

M., female, 18 *years.* The left incisors and the left canine were delayed in eruption, and were irregular and crowded. Much regulating has been done to the teeth and the first bicuspid has been removed in order to allow the teeth in front to come into line.

Nasal examination. The premaxillary wing on the left is slightly enlarged with a slight deviation of the septum into the right side. The axis of the deviation is horizontal. There is moderate enlargement of the wing of the premaxilla on the right running back one-half inch. Half way back on the vomer-ethmoid junction there is a small sharp spur. The arch of the palate is normal. Both halves are of equal height. There is slight chronic rhinitis, and a moderate granular pharyngitis.

M., male, 9 *years.* All the upper incisors of the first dentition are still in place. Normally these should have erupted two years ago. The incisor teeth are regular in outline but are movable on pressure as if they were on the point of being cast off. The nasal bones are in good line. There is no history of trauma. There is no nasal obstruction. The arch of the palate is normal and both halves are of equal height.

Nasal examination. Large vomer spur is on the left, running well back. The left premaxillary wing is much enlarged, the right premaxillary wing slightly enlarged. The septum deviate into the right nostril. The axis of the deviation is horizontal.

These four cases are from families in good circumstances so that the teeth have had every chance. In all, the line of the nasal bones was straight. In all, careful questioning of the parents brought out no history of trauma. In the first three cases there was moderate delay and irregularity in the eruption of the incisor teeth. In these examination showed an enlargement of the premaxillary wing on the side of the delayed tooth and a slight deviation of the anterior part of the septum. In the fourth case the first teeth were retained two years beyond the time when they should be shed normally. In this case there was a very large premaxillary wing on one side with a large vomer spur running well back from it, a moderate enlargement of the other, and in addition a deviation of the septum into the opposite side. So marked was the deviation that any increase from continued growth along the present vicious lines or any sudden increase from trauma must produce obstruction.

The case of the girl of eighteen is interesting because she started with delayed and crowded teeth. Her teeth were carefully regulated, however, and brought into line so that her septum escaped with but slight deformity. The lesson of such a case is obvious. At the time that I saw the second of these four cases both central incisors were equally and fully erupted and in good line but the history card showed that the left middle incisor came down six months after the right middle incisor and six months delayed. In the nose, on the side of the lagging incisor, examination showed that the premaxillary wing was enlarged and that there was a small vomer spur on the same side. The septum deviated slightly to the right. From these cases it is seen that the history of the teeth is to be read in the nose.

The results of the findings in the rest of the forty cases can be put very briefly. They are as follows: Wherever there was moderate and equal delay in the eruption of the central incisors the premaxillary wings were symmetrically enlarged at the floor of the nose on both sides. In such cases there was usually little if any deviation of the septum and there was no vomer spur. Where, however, there was marked inequality and delay in the eruption of one central incisor as compared with the other central incisor, on the side of the backward tooth the premaxillary wing was much enlarged or displaced and the quadrangular cartilage tipped out of its bed along the vomer ethmoid suture. As a rule the long axis

of the deviation was antero-posterior, roughly paralleling the spur, and the upper part of the cartilage was bowed toward the spur. Accompanying this horizontal deviation there was often a certain amount of sigmoid deviation, the axis of which was vertical. It is fairly common to find the root of the lateral incisor mounting into the outer part of the floor of the nose. Even when the lateral incisors are crowded out of the dental curve and are placed directly behind the central incisors, a deformity which is striking and one from which you would expect much at first glance, they seem to have but little influence on the septum as compared with the central incisors.

The findings of these forty clinical cases are borne out and the details of their formation explained, by the dissection of twelve

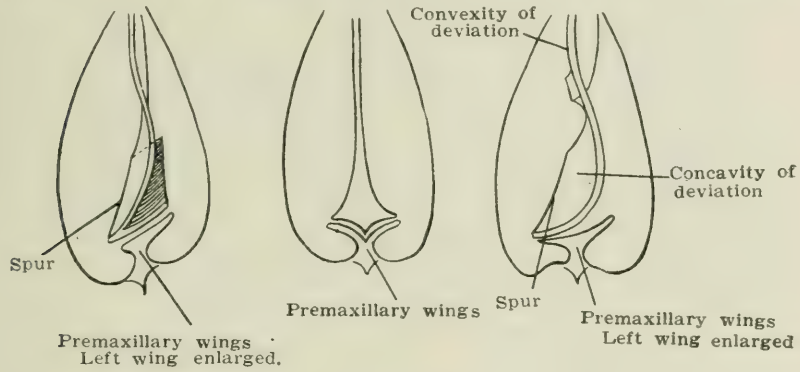


Fig. 1. Fig. 2. Fig. 3.

PLATE 14.

Diagramatic drawings of the types of deviation due to deformity of the premaxillary wings.

Fig. 2. Both premaxillary wings turned outward, causing a double basal spur of the septum.

cases in which an enlarged or displaced premaxillary wing caused a deviation of the septum. These specimens showed that if the pressure caused by the premaxillary wing is applied to the septum directly in its vertical axis, the lower edge of the cartilage stays in its bed but that the lower part of it folds sharply upon itself, the axis of the fold paralleling the upper border of the vomer. (Plate 14.) More often, however, the force seems to be applied a little to one side of the vertical axis of the septum. When this happens, the lower rim of the cartilage is forced out of its groove between the retaining premaxillary wings and the two leaves of the vomer and curls upward and outward. As it does this it breaks off

one premaxillary wing or stunts its growth and does the same thing to one leaf of the vomer groove. This results in the familiar spur along the vomer ethmoid articulation. (Plate 15.) The concavity of the lower portion of the cartilage about half way up the septum merges into a compensatory convexity. These dissected specimens bear out the clinical findings that the convexity generally is toward the spur and away from the enlarged premaxillary wing. (Plate 16.) Occasionally the reverse of this is found. The spur is on the side of the convexity and seems to crown it. The enlarged premaxillary

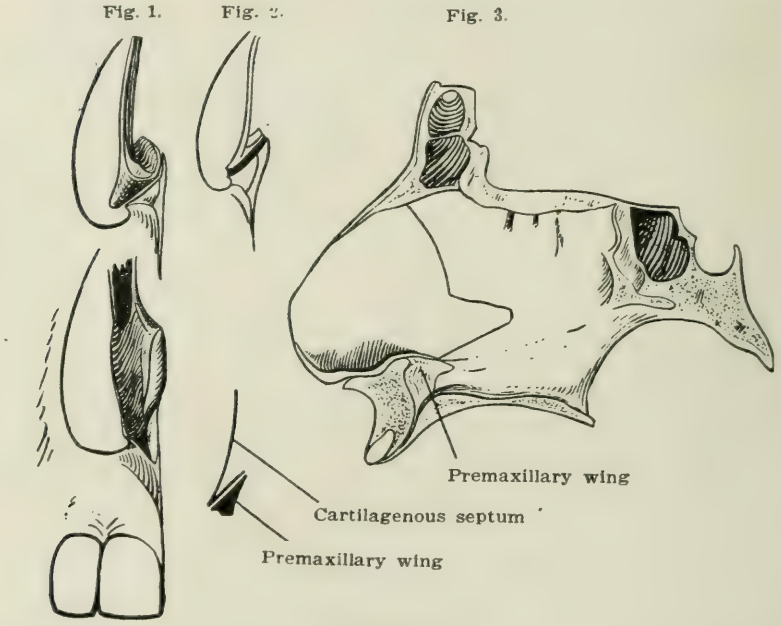


PLATE 15.

Specimen of deviation of the septum due to enlarged left premaxillary wing. Fig. 1. Dissection of the groove of the premaxillary wing. Left wing enlarged. Fig. 2. Scheme of the deviation.

wing is found in the dissected specimen to make an anterior basal spur which runs for a quarter of an inch to an inch backward. Further, when the two premaxillary wings are enlarged but slightly and are enlarged equally they often tip outward and make a small anterior basal spur of either side of the septum. Where, however, both premaxillary wings are evenly enlarged to a considerable extent and the pressure caused by this enlargement is not equalized by their turning outward, as in the case just described, the tip of the

septum is first pushed upward and then to one side. (Plate 17.) The septum in this case yields instead of the premaxillary wings. I found this condition in the skull of a three year old child.

The spur, as has just been said, usually is on the side of the convexity and often seems to crown it. The character of the spur naturally varies with its formation. Where the spur is made by a simple folding of the lower part of the cartilage upon itself the spur is all cartilage. On the other hand, where the cartilage breaks down one leaf of the vomer the spur would be cartilage above and

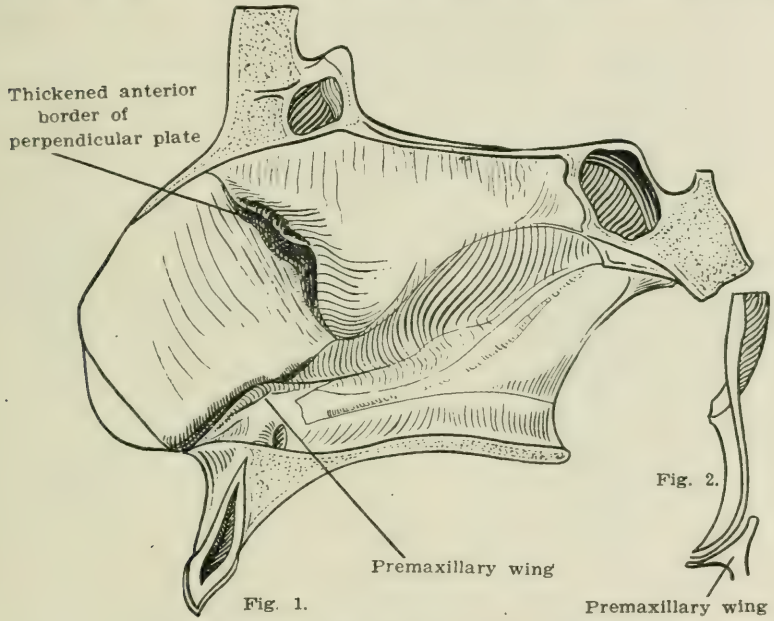


PLATE 16.

Fig. 1. Deviation due to enlargement of the premaxillary wing.
Fig. 2. Dissection of the deviation.

bone below. Where the vomer groove is distorted but not broken the spur would have a shell of bone above and below with a core of cartilage between. If one premaxillary wing is broken off or atrophies from pressure and the same thing happens to the corresponding leaf of the vomer the spur would again be all cartilage. It is common in the clinic to see a vomer spur end about two-thirds the way back on the septum in a sharp spine like projection. A dissection of such a case shows that this formation is due to the fact that the two leaves made by the lower edge of the perpendicu-

lar plate of the ethmoid and the two more pronounced leaves of the upper border of the vomer turn half round until they coincide with the long axis of the septum and then bend sharply outward and burst open. (Plate 18.) This spine-like excrescence may be all bone and very hard or it may have a core of cartilage.

Enlargement of the nasal spine, as well as enlargement of the premaxillary wing, can cause spurs and deviations of the septum. Enlargement of the nasal spine does occur but it is less common to find examples of it in the dissecting room than it is to find enlargement of the premaxillary wing. It should be borne in mind

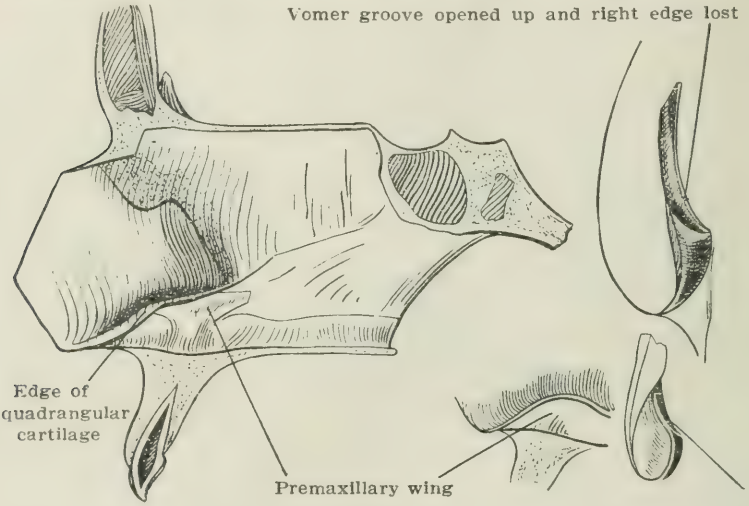


PLATE 17.

Deviation of the septum due to the enlargement of the left premaxillary wing. Small figures, the dissected anatomy of the deviation.

however ,that the nasal spine is the cause of deviations in certain cases. In the cleaned skull the nasal notch on the side of the enlarged premaxillary wing is higher than the notch of the other side. A good X-Ray will occasionally demonstrate this in the living.

I feel that good anatomical and clinical evidence has been given to show that enlargement of the premaxillary wings, the enlargement being due to delayed eruption of the incisor teeth, must be considered as important cause of deviations of the septum. There is yet more to be said about the teeth. Not only may the anterior openings of the nose be of unequal height and size, but also the posterior openings, the choanae. (Plate 19.) The anterior central

teeth produce the deformity in front and what might be called the posterior central teeth, the upper wisdom teeth, produce the deformity behind. The molar teeth and especially the wisdom teeth are brought into place by the downward growth of the tuberosity of the superior maxilla in which they are lodged. This shuts into place like a hinged box cover. At times it does not shut completely down leaving the wisdom teeth to erupt at a vicious angle. The palate bone which forms the outer side and the floor of the choana is fastened to the inner side of the tuberosity of the superior maxilla and must follow its movements. If, therefore, the normal down-

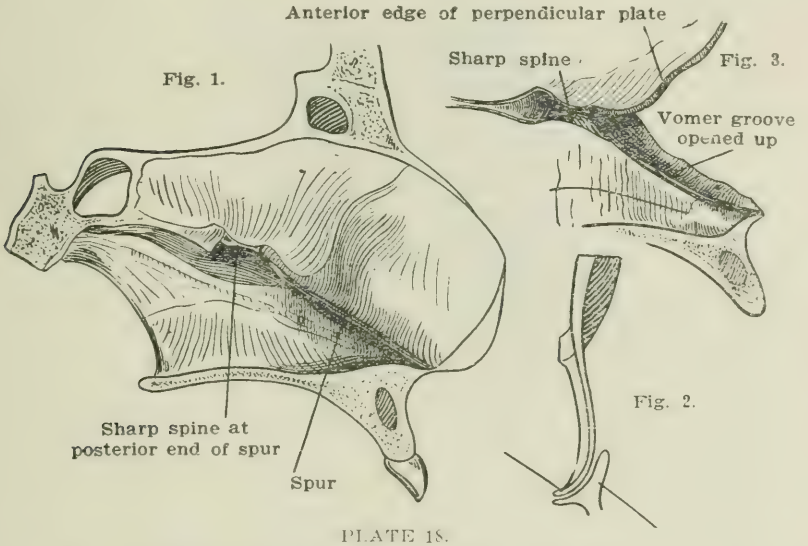


PLATE 18.

Deviation of the septum caused by enlargement of the left premaxillary wing. Fig. 2. Anatomy of the deviation and spur.

ward growth of the tuberosity, and in consequence the normal eruption of the wisdom teeth, is disturbed, this can cause an inequality of the nasal notches. When it occurs it probably plays a certain part in upsetting the equilibrium of the different parts of the septum.

In the four clinical cases which were given in detail I was careful to state that in all, the two halves of the arch of the palate were of equal height. I did this because there is a third cause of deviations of the septum which if it is connected with the teeth is connected with all the teeth of one side not as in the other cases with special teeth. The third cause is unequal descent of the antra. It results

in asymmetry of the whole of one-half of the palate. In such instances one-half of the palate is higher than the other half. Plate 20 illustrates this condition . The left nasal notch, the left choana, and the half of the palate between, are higher than the corresponding parts of the other side. This has caused hypertrophy and displacement of the left premaxillary wing and an opening up of the gutter made by them, and the displacement of the tip of the vomer toward the lower nasal notch. This inequality in the

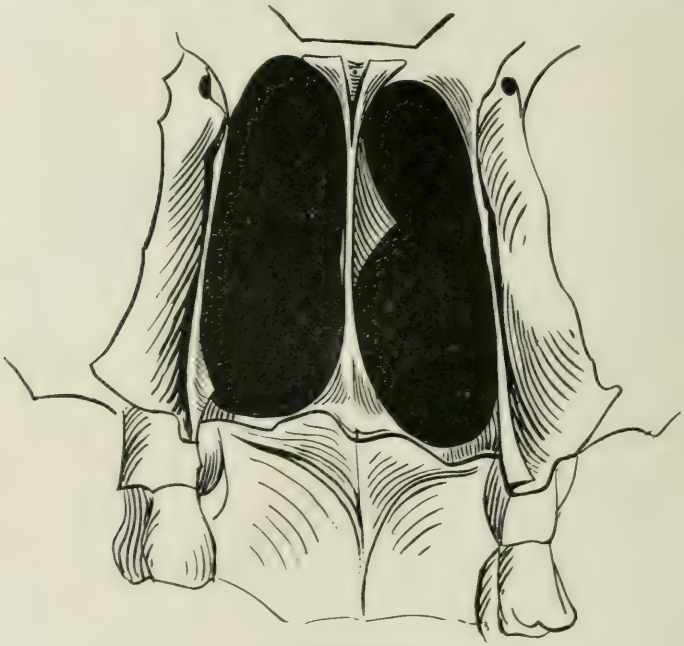


PLATE 19

Inequality of the choanae due to irregular descent of the antra. The left antrum has not descended as low as the right, so that the left choana is higher than the right choana.

halves of the palate is readily seen in the living by looking for it in the mouth. I found an example of this recently in a child of six years. Small differences in the antra are hard to make out in the X-ray plate, but in one of my adult cases the plate showed clearly that it was present.

When one nasal notch is higher than the other nature often attempts to equalize the deformity. For example, if the right nasal notch is higher than the left just the reverse of this may be true of the choanae. In these cases of inequality of the halves of the

palate the asymmetry may extend to the whole half of the head, but as a rule it is confined to the bones which make the hard palate and have to do with the teeth. Where, however, there is asymmetry of half of the head the nasal bones and the septum share in the general asymmetry. In such cases the cartilage of the nose both externally and internally follows the line of the deviated nasal bones.

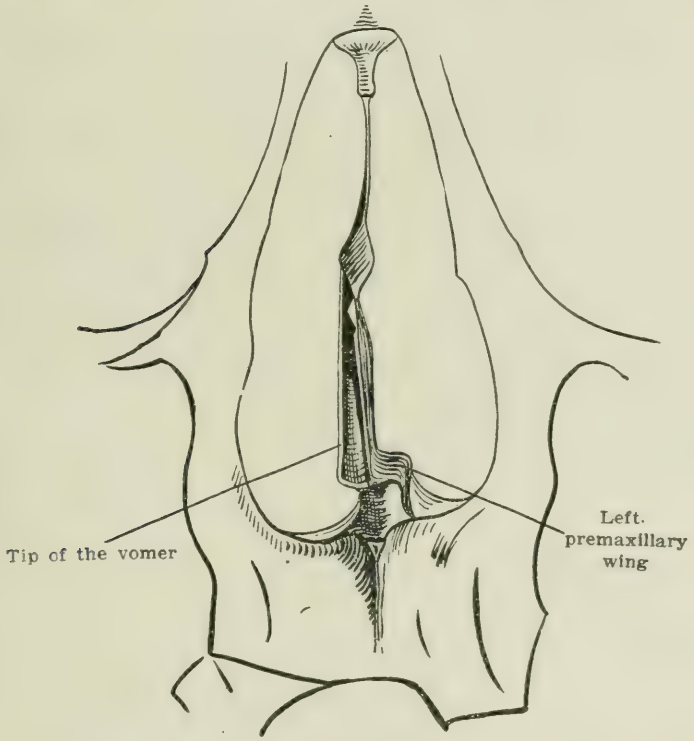


PLATE 20.

Specimen shown in Fig. 19 seen from the front. The left nasal notch is higher than the right. The left premaxillary wing is enlarged and has thrown the tip of the vomer to the right.

The last great cause of deviations of the septum is trauma. It used to be argued against trauma that the less civilized races, where there is the most chance for trauma, have straighter septa than the civilized races. This has been in great part disproved by the examination of prehistoric skulls. The negro may have a straighter septum than a white man. If he does it is probably due to the fact that the negro has the heavy premaxilla of the carnivora. The

resemblance is very striking. The bony vestibule of the nose of a negro is wide open and flaring and roofed with thick bone, just like, for example, the premaxilla of a bear. Owing to the strength of the premaxilla his septum is kept in good line.

The septum at birth for the greater part is cartilage. It is so resilient that one can study the effect of trauma upon it with ease. For the first year the articulations about the superior maxilla are so soft that it is possible by upward pressure in the mouth to collapse the face to a certain extent. It is somewhat startling to do this for the first time. By putting two fingers of each hand in a baby's mouth under the premaxillae and getting counter pressure upon the skull with the thumbs the face can be shut together very appreciably. This spring of the face must be a great safeguard during birth and early in infancy. I have often wondered how the septum fared at birth. By experimenting with a wet specimen of a baby's septum it is easy to see what must happen. During birth the nose is flattened with the face. In instrumental deliveries the flattening is extreme. In order for this to take place the septum folds upon itself vertically. After birth either the natural spring of the cartilage or the first wiping of the baby's nose straightens this deviation out. An instrumental delivery, however, might readily throw the septum out of its groove in the premaxillary wings or break one of the leaves of the vomer and so make the deviation permanent.

If pressure is applied to the baby's septum from below the cartilage folds horizontally the same as a playing card in the half closed palm. The septum is not ossified to its full extent until the eighth year. Up to this time, therefore, blows upon the septum, unless extreme, are probably taken care of by the spring of the cartilage. Great violence could either jump the tip of the vomer out of its bed in the premaxillary wings or fracture one or both of these wings and one or both of the leaves of the vomer.

After the perpendicular plate of the ethmoid has ossified all that it is to ossify its anterior border plays an important part in deviations caused by trauma. The under surface of the median border of each nasal bone is supported from above downwards usually for two-thirds of its extent by the superior border of the perpendicular plate of the ethmoid. The anterior border of the perpendicular plate of the ethmoid, therefore, starts from a point a third of the distance from the tip of the nasal bone to its root and

runs obliquely downward and backward to meet the upper border of the vomer just behind the premaxillary wings. (Plate 21.) The direction of the anterior border of the perpendicular plates of the ethmoid, however, is subjected to great variation. Instead of being roughly vertical as has just been described, it may run backward almost horizontally, in this way increasing the size or the caudal prolongation of the quadrangular cartilage, and turning the anterior inferior angle of the perpendicular plate of the ethmoid into a tongue-like projection.

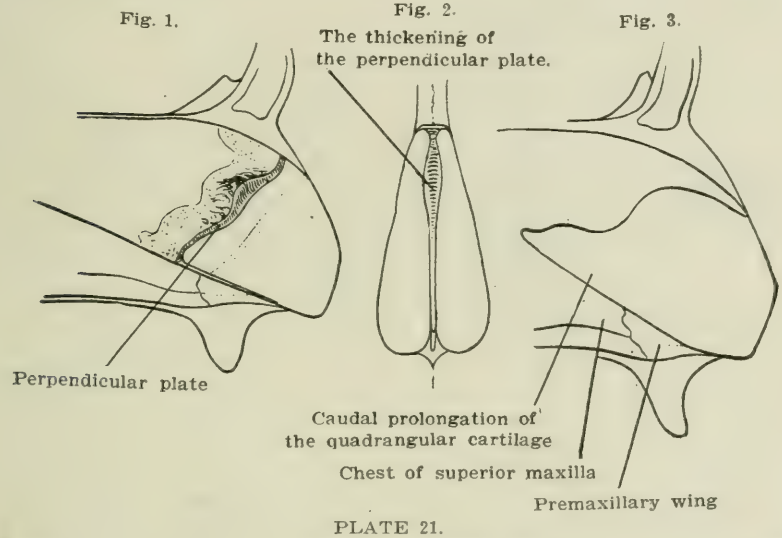


PLATE 21.

Fig. 1. A part of the quadrangular cartilage has been cut away in order to show the thickening of the anterior edge of the perpendicular plate of the ethmoid. This edge is ossified for some distance back.

Fig. 2. The thickening of the perpendicular plate.

Fig. 3. The anterior edge of the perpendicular plate runs horizontally backward and makes a large caudal prolongation.

Where the direction of the anterior border of the perpendicular plate of the ethmoid is practically vertical it is well fitted to withstand trauma. In such a case the force of a blow on the nose directly from the front is transmitted by the quadrangular cartilage squarely to this edge of the perpendicular plate and most of the force expands itself there. A hard blow produces a vertical deviation or folding of the quadrangular cartilage beginning below at the premaxillary wings and running upward roughly paralleling the anterior border of the perpendicular plate of the ethmoid. Experimental work on

the cadaver has proven that the articulation between the anterior border of the perpendicular plate of the ethmoid and the quadrangular cartilage is often too firm to be broken except by the greatest force. herefore the deviation parallels the synchondrosis but does not invole it.

Where the anterior border of the perpendicular plate of the ethmoid is vertical, it is adapted to withstand trauma, but where this border runs backward horizontally, it loses its function of a buffer. In this case it is not rightly placed to receive and withstand the effect of a blow from the front. The caudal prolongation directs

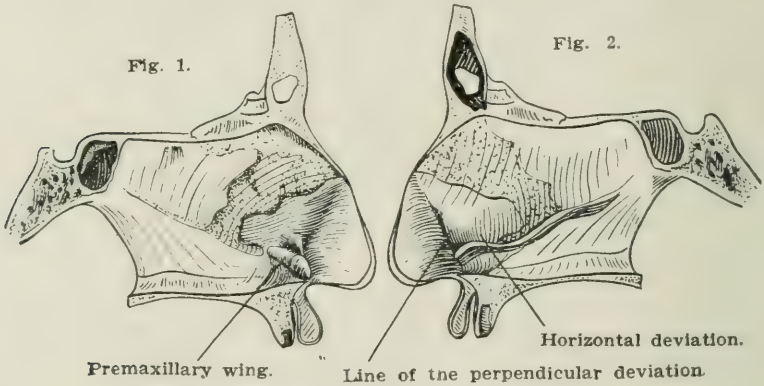


PLATE 22.

Plate showing a perpendicular deviation of the septum with enlargement of the right premaxillary wing. This type of deviation is characteristic of trauma. In this case, both the displacement of the premaxillary wing and the deviation were probably caused by trauma

the force beneath and beyond it, well into the middle of the septum. (Plate 21.) The tip of the vomer is forced from its bed between the premaxillary wings and one or both of these processes are broken. The caudal prolongation is driven as a wedge between the upper border of the vomer and the tongue-like projection of the anterior inferior angle of the perpendicular plate of the ethmoid. The anterior inferior angle of the perpendicular plate bends in consequence and bends or breaks one leaf of the vomer groove. The size of the caudal prolongation therefore, in great measure determines the form and the extent of the deviation produced by trauma.

The anterior edge of the perpendicular plate of the ethmoid is a natural buffer not only on account of its position but also on account of its thickness. The upper half of this edge is the thick-

est part of the septum. This thickening occurs on a level with the middle turbinate, and extends backward into the body of the ethmoid plate from a quarter to half an inch. Often this thickening is covered with exostoses. It would seem that these are probably due to the constantly repeated small injuries of childhood.

In short, a sharp anterior vertical deviation is characteristic of trauma. Such a deviation begins below at the premaxillary wings and runs obliquely upward paralleling the anterior edge of the perpendicular plate of the ethmoid. The perpendicular deviation almost always merges into a horizontal deviation which includes the anterior inferior angle of the perpendicular plate of the ethmoid. The shape of this part of the ethmoid plate materially influences the form of the deviation. (Plate 22.) This horizontal deviation generally turns to the same side as the vertical deviation.

Excessive trauma, applied either from the front or from the side, causes deviations and fractures of the septum which cannot be brought into any classification.

Treatment. I have nothing to say about treatment. That is not included in my subject. I wish, however, to add a word concerning prophylaxis. The obvious thing to do is not to leave the first incisors in too long. Dentists as a rule object to early extraction of the first teeth because they say that the second teeth erupt irregularly if the first teeth come out too soon. If this is so, there are two horns to the dilemma, namely, leave the first teeth in as long as possible and deform the septum or remove the first teeth early and deform the second, but keep the septum straight. Most men and all women would prefer straight teeth and a crooked septum to a straight septum and crooked teeth.

Just at present there is a good deal in dental literature about widening the arch of the palate and so widening the nasal fossae. The old fashioned split plate would undoubtedly accomplish this. It is yet to be demonstrated beyond a doubt that the modern methods accomplish more as a rule than a change in the angle of the teeth and an apparent widening of the arch. It is to be hoped that it will soon be proved that the nasal fossae can be widened by widening the arch of the palate. Because, if it can be brought about to any appreciable extent, the dentist can do much to prevent and to correct deformities of the septum. Dentists maintain that they can ensure by treatment the regular and normal erup-

tion of the incisor teeth. By accomplishing this they can save many a septum from deformity.

Summary.—A large number of deviations of the septum are caused by asymmetry in the development of the bones which make the hard palate. This inequality of development is usually due to delayed or irregular eruption of the incisor teeth, especially of the middle incisor. Delayed eruption of the teeth is caused in great measure by some disturbance of nutrition. When the eruption of one central incisor is sufficiently delayed it causes a deformity or hypertrophy of the premaxillary wing above it. This distorts the retaining groove made by the premaxillary wings. As a result, the septum slips from its bed in the vomer, and the groove made by two leaves of the vomer spreads open, one leaf or side of the V disappearing. This produces a spur along the upper edge of the vomer. As the cartilaginous part of the septum slips from its bed, the lower edge curls upward and outward, so that its lower portion becomes concave. Higher up on the septum this concavity gives place to a compensatory convexity. The convexity generally is toward the spur. On the side of the delayed tooth a short basal spur indicates the enlarged premaxillary wing. The upper wisdom tooth may deform the septum posteriorly. Occasionally deformity of the septum is caused by asymmetry of one half of the palate. This asymmetry shows in the nasal notches anteriorly and in the choanae posteriorly and in the mouth. Such extensive asymmetry is due to unequal descent of the antra. After the teeth are fully erupted and in good line there remains no evidence of the disturbance caused by their delayed eruption except in the nose.

Trauma as well as delayed eruption of the incisor teeth can displace the premaxillary wings and distort the vomer groove resulting in spurs and causing deviations anteriorly and posteriorly. The best explanation for the slight anterior deviations which are found so constantly is some fault in the eruption of the incisor teeth. Abundant dissecting room findings prove that deviations so started may extend far back on the septum and become obstructive.

DISCUSSION.

Dr. E. A. Bogue, of New York City, said that Dr. Mosher had pointed out with great care the facts of crooked septa and consequently of more or less occluded nasal passages, but admitted that, aside from extraction of the anterior teeth, he saw no cure for the

condition. Dr. Mosher had, however, himself indicated the remedy in an allusion in this paper. He had cited four cases, of which he said, "All of these four cases are from families in good circumstances, so that their teeth have had every chance." This, according to Dr. Bogue, did not always follow. He mentioned one family of seven children, in which one child had splendid teeth, beautifully arranged, good nasal passages and good palate vault. Another child of the same family had irregular teeth, deficient development, and from six to eight cavities in every tooth in her mouth. In fact the physical characteristics of all the children were distinctly different, and the teeth, being dermal tissues, had been affected by all that affects that tissue. When a child became a mouth-breather because of nasal stenosis, the tongue was withdrawn from its proper place in the roof of the mouth; and the pressure of the tongue, which is the formative influence in shaping the superior dental arch, was also withdrawn and lateral development ceased. Not only development of the dental arches, but of the pre-maxillae, vomer, ethmoid and palate bones as well, ceased. Extraction of teeth never helped development. It never made room, but on the contrary, it diminished the size of the dental arches, just as removing one or two staves would diminish the size of a barrel. It shortened the bite, it diminished the room needed for the tongue, and it interfered with the development of the palatine arch. If the extraction occurred in childhood it caused arrest in the development of the nasal and faucial bones, which could never thereafter attain, unaided, their normal size. If the extraction occurred on one side only, it might result in the eyes not being on the same level, so that one of the eyes sometimes remained undeveloped and defective. When Dr. Mosher spoke of the girl of eighteen who started with delayed, irregular and crowded teeth, and said that they were carefully regulated and brought into line, so that her septum escaped with but slight deformity, he had struck the key-note of the whole question. If, to begin with, breast feeding in the infant were promoted; if the child were kept in good health as it continued to grow; if it were given hard, fibrous food when its teeth began to appear, thus enabling it to so exercise the muscles of mastication as to promote the growth of the jaws, little of this retarded development so justly deplored by the essayist would be found. When found, however, it should be recognized that a mechanical obstruction to development exists. This obstruction should be removed by a mechanical enlargement of the temporary dental arches at the earliest proper age, thus promoting the development of the septum, the vomer and the vault of the palate, and so preventing irregularities in the arches of permanent teeth. By so doing not only would a far more effective and durable masticatory apparatus be provided, but a far more effective mechanism for speech and vocalization as well, the appearance would be improved, and a

more nearly normal septum and ample nasal passages would be attained.

Dr. J. H. Ingersoll, of Cleveland, Ohio, asked Dr. Mosher if he did not consider some of the bilateral projections from the septum as reversions to more primitive types. In many of the lower animals the septum has a bilateral projection partly subdividing each nasal fossa anteriorly. He believed that the spurs occurring along the union of the septal cartilage with the bone were rudiments of these structures.

Dr. Charles P. Grayson, of Philadelphia, so far from regretting that the paper did not give some information as to treatment, said that Dr. Mosher's work had made clear the causation of the condition, and at the same time he had suggested, not a method of cure, but what is much better, a method of prevention. He did not entirely agree with Dr. Mosher that dentists had for a long time been familiar with the relation between narrow alveoli with crowded teeth, and septal deformties. He had found a wide difference of opinion among dentists concerning this subject. Orthodentistry, however, had made rapid strides, and it would be well to form a closer alliance with the dental profession.

Dr. Mosher, in closing the discussion, said that treatment along the line of spreading the teeth would accomplish a great deal. Dr. Ingersoll's suggestion with reference to reversion was probably true.

PAPER:

A REPORT OF A CASE OF FIBRO-MYXOMA, INVOLVING THE LEFT SUPERIOR MAXILLA, INCLUDING THE ALVEOLUS, FLOOR, ANTERIOR AND POSTERIOR WALLS, AND OF SEVEN YEARS' DURATION.

W. H. HASKIN, M. D.

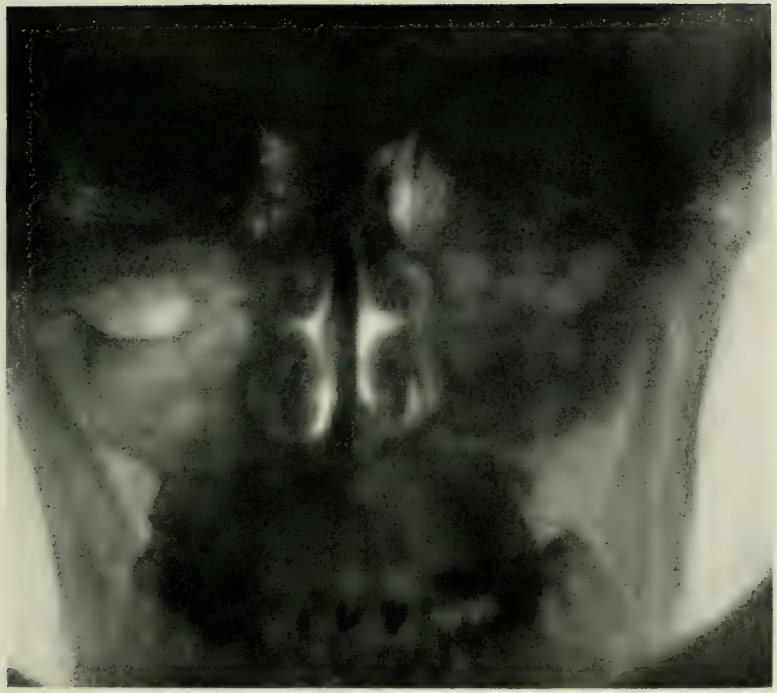
The knowledge of the life history of this peculiar form of growth appears to be very limited, and after considerable research I was unable to find any literature to assist me in the care of this patient. It seems to me that the duration and the persistent recurrence after repeated attempts at removal with curette and cautery, and the fact that it could not be classified as an encapsulated tumor, for it ramified in all directions and completely absorbed the bone in many places, point to a much more serious condition than is generally accepted for a fibroma. At the time of my operation there was decidedly more myxoma present than fibroma, and judging from the history given by the patient I believe that has always been the case. The freedom from pain, the absence of any tendency to suppurate (the surface always healing over after previous operations), and the lack of any lymphatic involvement contra-indicate malignancy; but, notwithstanding these facts, the ramifying nature, the recurrence and the gradual loss of weight of the patient seem to indicate a serious condition, the true nature of which we have not yet grasped; and it is with the hope of learning more of its nature that I present this, to me, most interesting case.

The patient, Miss H. M. H., was brought to my office on February 2nd, 1907, by Dr. A. F. Bauer, a former student of mine at the Dental College, with the following history: Family history excellent, both parents and two brothers being alive and in good health. Several years ago she suffered with menorrhagia and was cured by currettement. With that exception her history was good, and she had never been ill to her knowledge.

The following letter is the patient's own description of her case: "About the Fall of 1900, while having some dental work done by

Dr. F., of New York, I called his attention to a wisdom tooth that was troubling me. He lanced it, and later, I believe, he lanced it a second time. In February, 1901, I consulted a dental surgeon of Brooklyn in regard to having a tooth straightened. He at once noticed a small enlargement over or near the wisdom tooth, and called my attention to it, saying it ought to be attended to. I believe he then removed the tooth. Upon my return home I saw our family physician of Newark, in whose hands we placed the matter for treatment. In February, 1901, he sent some of the growth to the Board of Health of Newark, N. J. They pronounced it to be ————————? and not malignant. The physician continued his treatment of it, cutting out at times considerable masses of it, hoping thus to check or destroy the growth. For a time it did not seem to be enlarging, and it caused no discomfort, so little attention was paid to it. But in April, 1904, it became worse and was again treated when it was necessary to remove another tooth. From that date until February, 1907, it was not treated.

For two years nothing had been done, and as there was no pain the patient did not realize the serious nature of the condition until she consulted her dentist again, who immediately advised her to see a surgeon and referred her to me. For some months she had been unable to masticate on the left side, and occasionally had some dull aching on that side, but otherwise was not inconvenienced. She had at times noticed some puffiness under the left eye, but close questioning did not elicit any history of nasal discharge at any time. There had been a slowly developing mass appearing under the malar bone, but not until recently had it caused the left side of the face to appear fuller than the right. As said before, there was no actual pain, but at times there had been some dull aching on that side. From without the tumour felt hard and unyielding. Examination of the mouth revealed a great thickening of the alveolus from which the last two molar teeth had been extracted. The thickening extended forward to the canine tooth and inwardly over the palate process to within one-half an inch of the median line. Externally it bulged out forming a tumour the size of half an English walnut, with a hard bone-like wall, which extended upwards to and apparently involved the malar process itself. Along the free border of the alveolus the mass felt soft and fluctuating and appeared somewhat translucent. The first molar and the second bicuspid were very loose in their sockets owing to the absorp-



tion of the alveolar bone tissues. An X-ray photograph shows this absorption very plainly. The nose was found to be in perfect condition and also the throat. Transillumination was negative, both antra showing light. On February 11th, 1907, under cocaine and adrenalin anaesthesia administered hypodermically, I made an incision along the free border of the alveolus, expecting to find a cyst. I found the fluctuation was due to the presence of a large amount of a semi-solid gelatinous mass, grayish pink in color, and I removed some of this for microscopical examination. The molar was then extracted and a probe was readily passed into the antrum through a root, it being found full of a soft growth. There was very little hemorrhage. The wound was packed with iodoform gauze, and the patient returned to her room to await the report of the pathologist. This was received on the 14th, and was as follows: Fibrous and epithelial hyperplasia in one piece of tissue submitted. In another piece there is a matrix of homogeneous groundwork taking haemotoxylin stain, and in it are a number of finely wandering embryonic connective tissue cells. Diagnosis:—Fibro-myxoma.—Dr. J. Wright, Pathologist.

On the strength of this report I advised complete removal and on February 26th this was done at the Manhattan Hospital, with the assistance of Drs. H. Smith and G. H. Ward. The operation was performed through the mouth, the floor, anterior wall and most of the posterior being removed in reaching the limit of the growth. It extended into the apex of the antrum, but in this region it was not attached, seeming to shell out rather easily, and the roof and inner wall of the antrum did not appear to be involved in the growth. Having dissected the periosteum from both the anterior wall and the palate process, these were removed together with the entire alveolar process up to the lateral incisor. The greater part of the palate bone was removed exposing the muscles and opening the zygomatic and even the pterygoid-maxillary fossae, the delicate fatty tissue protruding into the wound in these regions. Having avoided the tuberosity and the posterior wall until the last, on account of the palatine arteries, there was but little hemorrhage, the growth itself being singularly non-vascular. The involved bone was very soft, as though absorbed, and was easily removed with curettes. The growth also seemed to shell out in large masses, especially where the myxomatous tissue predominated. The greatest difficulty was found at the tuberosity and along the posterior wall, but

this was overcome by using a large, powerful, round fenestrated tonsil punch, of German make, which cut through the softened osseous tissue with ease. In all, I should say, the growth weighed between two and three ounces, though possibly more. No opening was made into the nose or the orbital cavity, and fortunately the soft palate was not involved, so that there was no opening in that region. The cavity was packed with iodoform gauze, no attempt being made to introduce sutures. Dr. Wright reported again that the growth was fibrous-myxoma, with no evidence of sarcoma.

Subsequent history: Uneventful. Three months after operation the extensive cavity had entirely closed with apparently healthy tissue. The patient had gained in weight, and was being fitted with an endenture.

The literature on growths of this nature is exceedingly scant, as said before, being merely mentioned in the classification of tumours in most text books. The American Encyclopedia of Medicine says about them: "Fibrous-myxoma usually occur in the region of the alveolus. When arising from the alveolus adjacent to the antrum it may project into that cavity and appear to be growing from it. These fibromata are considered by Bland Sutton to be of the same origin as odontomata. Some people consider that they may result from chronic inflammation around the apex of a tooth." Garretson, Marshall and Grant, in their books on the surgical diseases of the jaws,—books that are used as text books in the Dental Colleges, make no mention of these growths. Dr. Wright has written extensively on nasal growths which have been generally regarded as of this nature. Posey and Wright do not mention it. Kyle mentions it as appearing in the nose.

The most extensive description that I could find, however, is in The American Practice of Surgery, by Drs. Bryant and Buck:

"Myxomata are tumours of mucoid character. Structurally they are composed of cells floating in a homogeneous, semi-fluid mucinous matrix. These cells are mononuclear, bipolar or stellate, and provided with more or less elongated protoplasmic processes which interlace freely. The intercellar substance varies in amount in different tumours and in different parts of the same tumour. When abundant, it gives a characteristic gelatinous, semi-fluid, somewhat translucent appearance to the growth. Myxomata are grayish, or pinkish gray in color, owing to the presence of blood vessels that are more or less distinctly visible in the substance. On section, a

jelly-like or ropy substance—mucin—exudes, which is not soluble in water, and gives a whitish precipitate with alcohol or dilute acetic acid.

Myxomata are rarely pure in type, but are usually combined with other tissues of a homologous nature. Mucoid tissue is closely related to fibrous tissue. The truth of this is evident when we remember that in the foetus the fatty tissues and fibrous tissues are first blocked out in mucoid material. Consequently, myxomata are commonly met with in the same regions from which fibromata and lipomata also spring. Further, mucoid tissue occurs in the adult body only in the vitreous humor of the eye, from which structure myxomata never develop.

Myxomata, therefore, always exhibit a certain deviation from the tissues in which they are found. In other words they are heterologous within narrow limits. The embryonic character of the cells also explains the well known instability of the tumour, which, though classed among the benign growths, has a tendency to take on malignant action, the transformation occurring in the direction of the sarcoma.

True myxomata have to be distinguished on the one hand from tumours, such as fibromata, sarcomata and carcinomata, that have undergone secondary mucinous degenerations; and on the other, from growths that, owing to vascular disturbance, have become oedematous. Myxomata (fibro and lipo) may originate in the connective tissue of the skin, fascia, periosteum, mucous membranes, and muscle sheaths; in the subcutaneous and subserous fat, the bone marrow, and occasionally, in the mamma, salivary glands and testis.

Myxomata of the mucous membranes occur singly or as multiple primary tumours. The ordinary mucous polyp of the nose is a good example of these growths. Myxo-sarcomata, so called, are due either to a cellular transformation of a simple fibro-myxoma, or to the mucinous degeneration of a sarcoma. They behave as sarcomata and form metastases."

This last account, as will be seen, gives an excellent description from the laboratory standpoint, but does not deal with the history of the growth, its effect upon the general health of the patient, the prognosis, or the best method of dealing with it. In this case we have a tumour of at least seven years duration, practically painless with no glandular enlargement, but extending out in many directions, apparently non-encapsulated, and destroying the bone

tissues which it invaded very much as would a malignant growth. Again it has repeatedly healed after curettement and has never shown any tendency to ulcerate, both of which facts rather disprove the thought of malignancy. Dr. Wright's report, having been made after examination of several pieces of the growth, also indicates the benign nature; and yet the question is, where can we draw the line between benign and malignant tumours? It seems to me that any growth that can so effectually absorb bone tissue, that returns repeatedly after operation (the thoroughness of which, however, I cannot vouch for), that appears to be without capsule and ramifies in bone, muscle, open space and fibrous tissue alike, does not belong in the benign class, and should be considered more seriously than has been done up to the present time. Bland Sutton's theory of a pericementitis being the cause or the starting point may seem to be borne out in this case, as its history of wisdom toothache preceded the growth. Dr. Cramer first noticed it while filling a cavity elsewhere and advised that it be attended to. If there is no recurrence after this extensive removal, it will be very easy to fit an obturator over the remaining cavity, with attachments to the opposite side, and with a complement of teeth that will give use for mastication, and at the same time will support the cheek if there should be much sinking in.

DISCUSSION.

Dr. J. Price-Brown, of Toronto, Canada, reported a similar case in which he had recently operated, first, by removing a large section of the anterior wall of the antrum in the region of the canine fossa, then taking out a considerable portion of the growth with the forceps, and dissecting out the balance of the mass. The attachment was entirely to the septum, and the tumor shelled out complete. It weighed about two ounces. The pain and difficulty were relieved and the patient was in a very fair condition. The question had arisen as to whether it were possible for this fibromyxoma to develop into sarcoma. The pathological specimen was presented to this society at the opening of the morning session.

Dr. Talbot R. Chambers, of Jersey City, N. J., reported a case which had come under his observation three months previously, in which there was considerable pus. A tube was introduced at the outer canthus, one at the corner of the mouth, and another at the angle of the jaw. The microscopic report was angio-sarcoma. Being unwilling to remove the entire side of the face, the patient was allowed to go. He returned later with the process apparently entirely quiescent. The microscpoe did not always tell exactly what was expected. A myxoma might be malignant and it might be non-malignant.

PAPER:

THE TREATMENT OF FOREIGN BODIES IN THE RESPIRATORY TRACT AND ESOPHAGUS.

PROF. G. KILLIAN.

GENTLEMEN: The invitation of your President to deliver an address on the treatment of foreign bodies in the air-passages and œsophagus is accepted with great pleasure, inasmuch as it relates to a method of examination in behalf of which I have, for the past ten years, been making an earnest propaganda, in the desire to benefit the afflicted by its general dissemination.

RESPIRATORY TRACT.

I need not say much with reference to the upper respiratory tract. We are often called upon to treat foreign bodies in the nasal cavities, especially in children. As a rule, they are buttons, beads, etc., made out of various materials, cherryseeds, beans, peas, and occasionally a forgotten cotton tampon. Nasal calculi also belong to this class.

Once I was called upon to remove a free osteoma, of the size of a hazel-nut, which had been left in the nasal cavity after an operation performed the year before, and which rolled to and fro with the movement of the head. It was incomprehensible how the patient could endure the condition so long. Another case was that of a child who accidentally ran a splinter into his nose. Another infant was brought to me suffering from very high fever, bad smelling nasal discharge, and swollen glands, resulting from a paper plug which his sister had stuck into his nose some time before. That the severe clinical picture depended upon this was shown by its disappearance after removal. Finally I must mention a case in which a wooden plug, as thick and long as the little finger, was shot through the maxillary sinus, passing into the nose and perforating the septum.

Foreign bodies in the nasal cavities in children are generally found in the vicinity of the vestibule, if no unskilled efforts at removal have been made. In one of my cases, the ethmoid bone

was penetrated as far as the orbit in the fruitless search for the foreign body. Inflammation symptoms may follow such efforts, which may make the discovery of a foreign body very difficult.

The simplest and the most common method of removal is to introduce a thick nasal probe, bent forward, beyond the foreign body, and then draw it from behind forward and remove it. The little patient often, involuntarily, assists in this by moving the head backwards. A strong pair of toothed forceps is the best instrument to use for large impacted foreign bodies. When necessary, a great amount of strength may be used with this instrument, so that the foreign body may be extracted with certainty.

So far as foreign bodies in the accessory cavities of the nose are concerned, the maxillary sinus is the principal site. They are generally trochars and drainage tubes which are shoved into artificial openings. Occasionally, roots of teeth or portions of bone, remaining after operation, are found. I have often succeeded in locating and extracting a drainage tube through an alveolar fistula by means of a nasal speculum. I have occasionally removed such objects while performing the radical operation.

Foreign bodies are seldom found in the epipharynx. I was once called upon to remove a collar button which I had drawn out of the larynx, under narcosis, with the head hanging downwards; it slipped from me and fell into the epipharynx. Its removal was more difficult than I had anticipated.

In the mesopharynx and hypopharynx, fishbones are most frequent. They are found in the region of the tonsils, sometimes at the base of the tongue, and rarely in deeper positions. They are readily removed with forceps. Pieces of bone may be lodged in the sinus pyriformis. I was called upon to remove a splinter of bone, sharp at both ends, which was stuck in this place, causing the patient to have severe symptoms, though it was not discovered by the physician in charge. In another case, deep pharyngeal and laryngeal abscesses, resulting from a chicken-bone sticking in the sinus pyriformis, were spontaneously evacuated and cured.

Foreign bodies in the larynx constitute an extensive and interesting chapter of our subject, but are not so common as generally supposed. As a rule the foreign body immediately travels further down. Sharp and uneven objects are most apt to be caught in the larynx; needles, fishbones, bone splinters, shell of nuts. Once I saw half of a dental plate lying there.

The laryngoscopic method of extraction has, in this particular, achieved a great triumph. It may be called the normal method for adults, as long as the foreign body is not firmly wedged in. In children, the removal through the agency of the laryngoscope is very difficult, and though it may be successful under narcosis, still our old time technic has its limitations. It entirely fails when the foreign body is tightly wedged in. The smaller the child, the greater the difficulty. It was formerly necessary to take refuge in laryngofissure, but now we have the additional advantage of direct laryngoscopy, which makes laryngofissure superfluous in the majority of cases. In all doubtful cases in children, I should recommend the practice of entering directly with the tube, under narcosis, and with the head hanging down.

The number of cases treated so far in this way is small (Denker, Garel, Guisez, De Stella, G. Killian), but it justifies the greatest hope for the future. I recommend you to use my tipped tube spatula. The base of the tongue is drawn forward with it and the epiglottis and inner surface of the larynx are cocainized. Under narcosis, this is done with the head hanging down. The spatula is then passed over the epiglottis which is thus drawn forward. By suitable illumination with an electric forehead lamp (Kirstein's) or with an electric extension lamp, a beautiful view into the larynx is obtained not only as far as the vocal bands, but also into the subglottic space and deeper, provided the local and general anesthesia is sufficient. Foreign bodies can be readily seen and removed with suitable forceps. If the process is made difficult by the mucus that collects, this is removed by my pump (which has lately been modified by my assistant, Bruenings). Anyone who has had experience can, under certain conditions, avoid tracheotomy, in recent cases, if the danger of suffocation is not too great, by rapid performance of direct laryngoscopy, as Denker succeeded in doing in one case.

FOREIGN BODIES IN THE TRACHEA AND BRONCHI.

I come now to the treatment of foreign bodies in the trachea and bronchi. In these, the direct method has the field entirely. The results have been extraordinarily successful. Since 1897, when I reported my first case in the Muenchener med. Wochenschrift, until the end of March this year, 164 cases have been reported. (I count only the cases in which a foreign body was actually present.) To my certain knowledge, however, there are a

large number of cases still unreported, so that altogether there are 200 or more. Practitioners have made themselves familiar with the new method in most civilized lands. It was very early and ardently employed in the United States.

In order to reach a foreign body in the trachea or bronchi, it is necessary to introduce a tube of proper caliber and proper length through the glottis, that is, to perform upper direct tracheo-bronchoscopy.

The tube should be chosen just wide enough to pass smoothly through the larynx. In adults, its caliber should be from 9 to 14 mm., and in children, as a rule, it must be reduced to 7 mm. Very small children require tubes from 5 mm. or less in diameter. The length of the tube should correspond, in adults from 30 to 40 cm., in children from 20 to 30 cm., or even less. The actual measurement for the removal is to be taken from the upper row of teeth to the point where the foreign body lies, with the head bent back. This is easily ascertained before the operation.

The variations in the age of patients, the size of their bodies, the width of the larynges, and the location of the foreign bodies are so great that a great many different tubes must be kept on hand. Furthermore, it sometimes happens that a change from a longer or shorter tube is necessary during the operation. In order to secure a desirable simplicity in this particular, I have effected the lengthening of the tube by introducing secondary tubes. The sliding tubes of Bruenings, which I now show, have been constructed accordingly and are very efficient for this purpose. They are composed essentially of a tube spatula, through which a second tube may be pushed as far as desired. An equipment of five or six of these tubes is sufficient for all adult cases. A regulator, attached to the lateral wall of the tube spatula, controls the introduction of the tube. The tube spatula is readily inserted and passed through the glottis. We can easily introduce the sliding tube any distance from the middle of the trachea to the bronchi of the lower lobes. Two lateral openings in the sliding tube renders respiration easier. They are especially necessary when it is desired to introduce a tube, filling the larynx, into a branch bronchus, where the lung tissue is not sufficient for the purpose of respiration (shut off by foreign body or tissue changes).

Good local and general anesthesia must be secured in order to introduce such a tube into the deeper air passages. A hypodermic

of 0.01 to 0.02 grams of morphine is given to adults a half hour before; in children, the internal administration of codein, five to fifteen drops of a one per cent solution, according to age, will be advantageous.

When no narcosis is to be employed, the larynx and trachea are cocainized by the aid of a mirror while the patient is in the sitting position. In this connection, the epiglottis may be pulled so far forward that a cocain applicator, extended perpendicularly, introduced through the mouth, with the aid of a mirror, will glide over the posterior wall of the larynx into the deeper parts. This procedure may also be undertaken by direct laryngoscopy or by the aid of the finger. Wherever I use general narcosis, I cocainize only after it is sufficiently deep. Then the mouth speculum is introduced, the tongue seized with the forceps and pulled forward and cocainization is made by means of the tipped tube spatula, as before stated.

It is best, in performing direct bronchoscopy for the removal of foreign bodies, to have the patient in the recumbent position, whether or not a general anesthetic is used. In this way all the disturbances, so easily called forth by the accumulated saliva and mucus, are suppressed.

The patient should be placed so that the foreign bodies lie at the highest level and the head at the lowest, since in this way the clearest field of view is obtained. Generally, I have the patient lie on his back, but lately I have begun to place him on his side, which seems to be the most advantageous position.

It is a great mistake to bend the head too far back at the introduction of the tube spatula. One should begin with a moderate bending of the head backwards and increase this during the procedure as far as occasion demands. The tube spatula is introduced through the angle of the mouth from the opposite side, if the situation of the foreign body is known. The epiglottis, arytenoids and vocal bands are sought and the instrument is then passed between them into the trachea. If the cocainization has been sufficient, there will be no interference at the glottis.

After the deeper portions and the bronchus especially have been carefully cocainized (without pushing the foreign body deeper), the sliding tube may be introduced first to the bifurcation and then into the affected bronchus. It should be advanced until it reaches the neighborhood of the foreign body which is often covered with

mucus and granulations or but slightly visible. It is then necessary to suck up the secretion with the pump. The granulations must be carefully treated so that no hemorrhage from in front occurs.

If the foreign body does not lie in a position where it may be easily removed, the neighboring mucous membrane should be made anesthetic by the application of cocain on a small cotton carrier or by a cocain spray (weak solution) inasmuch as this region is very hyperesthetic. The slightest movement of the foreign body causes severe paroxysms of coughing. The greatest difficulty results from neglecting to cocainize in this way, for it is clear that everything else has been made anesthetic, especially the whole passageway to the foreign body, except the mucous membrane directly adjoining.

The procedure is much simpler in tracheotomized patients whether tracheotomy has already been performed or whether we ourselves have been forced to perform it on account of the severe dyspnea, or must perform it because the extraction cannot be accomplished by the upper method.

Lower direct tracheo-bronchoscopy seldom requires general anesthesia. Simple cocainization of the tracheo-bronchial mucosa is sufficient. Larger and shorter tubes may be used. For the reason already stated, it is best to have the patient in the recumbent position. As a rule, Bruening's new instruments are the best adapted for lower tracheo-bronchoscopy.

The extraction of foreign bodies is a subject in itself. No general rule can be laid down, but each case is a law unto itself. It depends upon the character of the foreign body, its position and the condition of the neighboring structures. The whole mechanism of extraction often requires thorough deliberation and experience is most advantageous.

I have thought that it would be of most service to you if I gave you the results not only of my own methods, but also those of others, and even those of prebronchoscopic times. I have, therefore, thoroughly studied the literature and I give you the results condensed.

We will pursue the subject better if we divide foreign bodies into groups and establish the best and most certain methods of extraction for each. In this way the novice may quickly obtain the most complete information regarding a given case and may guard himself against serious mistakes. Above all, it will be of

value to him to accustom himself to the proper use of the most reliable instruments for extraction.

Foreign bodies are best divided into two groups from the standpoint of the extraction (this is practical and most important).

I. Hard.

II. Not hard.

HARD BODIES.

The hard variety have in common a definite size, form and surface quality. I first separate those which extend mainly in one direction: Needles, fishbones, nails, screws, and steel pens. Then follow those which are extended in two dimensions: Flat bodies like coins, pebbles.

Bodies with three dimensions: with smooth surface such as beads, cherry-stones, coffee-beans; those with uneven surface and contour, as prune-stones, pieces of bone, etc.; those with irregular form such as teeth, pieces of metal, metallic objects of different types but typical form, and collar buttons.

Hollow bodies of different materials and of the widest variety constitute a separate group.

BODIES NOT HARD.

Bodies with consistency from wood to cork have an intermediate place. Fruit seeds, which are easily broken, are placed in an individual class, likewise the semi-soft vegetable seeds. Then come the really soft bodies, which must be considered individually, for instance, cereal spikes and especially seeds which are likely to swell (beans, etc.).

Needles.—Sprengel, 1; G. Killian, 2; Garel, 1; Ingals, 2; Coolidge, 2; Gottstein, 1; Lombard, 1; Elsberg, 1; Brokaw, 1—12 cases.

Those which were inspired were usually of the sewingneedle variety, though in one instance it was a Pravaz syringe needle. The patients, as a rule, are adults, which makes the removal much easier. As needles cause only slight pulmonary symptoms, there is no necessity for immediate extraction, so that if success does not attend first efforts there is no special danger in waiting and the necessary procedure may be repeated.

It is a very valuable circumstance that the Roentgen picture is usually positive, showing the exact site and position of the needle. We can, as a rule, be certain that the head of the needle lies downward and that the point sticks upward and sidewise. Its fixation,

as a rule, is only slight. The point does not simply stick into the mucosa; it is usually surrounded by granulations or has caused a small abscess. As there are only slight disturbances of the bronchial mucous membrane, the examination will not be made difficult by mucus; on the other hand, we must remember that there is a great hyperesthesia in the neighborhood of the needle which must be overcome by careful cocainization.

Needle cases are more suitable for upper bronchoscopy than any others, for the reasons given. Lower bronchoscopy is required only in extraordinary cases. Our report of cases gives no proper light as to the future, because tracheotomy has been too frequently performed, since upper bronchoscopy was not sufficiently trusted by operators.

My first case certainly constitutes an exception. I made a sincere effort to find the needle by upper bronchoscopy without result, and likewise lower bronchoscopy, although the X-ray picture was positive. Ten years had intervened since the needle had entered the trachea, and therefore it might have been imbedded anywhere in the lung tissue.

When the needle is brought into good view and the position of the point ascertained by means of the tube, it is easily grasped near its point by von Eicken's right angled short-jawed forceps and freed from the bronchial wall by a slight backward motion and then drawn outward through the tube. If the forceps slips off the smooth metal, it may be covered with a thin rubber pad. The needle may also be freed from the bronchial wall by pressure of the rim of the tube over the place where the needle has penetrated.

All the cases cited above were followed by good result; in but one case (Lombard) was the cure delayed by pneumonia.

Fishbones generally are caught higher up and seldom reach the trachea or bronchi. Only two cases were observed by means of the bronchoscope, one by my brother and the other by Gottstein. Both were in young children, 3½ and 1 year. In the first case, upper bronchoscopy, under general anesthesia, was of excellent service. The bone was readily removed with a hook and the child rapidly recovered. Tracheotomy was necessary in the second, on account of threatening suffocation. The bone, which was subglottic at the beginning, was found by means of lower direct tracheoscopy to be in the trachea, from which it was easily removed.

Nails. (Cases of Lindt, H. v. Schroetter, v. Mikulicz, Coolidge, Helferich, Neumeyer, Guisez.)

We have in these cases to act, in general, as with needles, as the conditions are exactly the same. The head lies, as in the other instance, generally below, easily demonstrated by the Roentgen picture. They are generally found deep in the right bronchus.

The patient is asked to show a nail similar to the one in the bronchus, for the purpose of orientation. As a rule, upper bronchoscopy and von Eicken's forceps will be successful. The latter has the value of drawing from the side so that one may see what is being done. Of course good results may be obtained with any suitable straight forceps. The electro-magnet is of use only when the nail is very small and deeply lodged, as happened in two cases. A rod shaped electro-magnet is necessary so as to permit its introduction deep into the tube.

Steel Pens (Cases of Borchardt and Gottstein) are to be treated in a similar way.

Coins (Cases of Killian-Worms, Coolidge, Guisez, 3) have been found in the deeper air passages only in adults. They are apparently too large for children and are therefore usually swallowed. Coins are flat bodies and lie usually diagonal to the bronchial lumen. The current of inspired and expired air apparently seeks to keep as broad a passageway as possible both from above and below by the side of the easily moved coin. Our extraction instruments in the form of forceps are particularly useful in drawing out diagonally placed flat bodies. It is therefore to be expected that good results will follow the use of upper bronchoscopy with proper technic, as was demonstrated in the cases above cited.

Pebbles. (Cases of G. Killian, von Eicken, Kahn, Pieniazek.) The inspiration of pebbles is a specialty of children from 2 to 6 years of age, who like to put such things in the mouth. They are partial to flat, smooth stones which require the same method of extraction as coins. Smooth stones present, however, greater difficulties. The pebbles selected by children are generally so large that they cannot fall deep into the bronchus, but remain at its entrance and often partially in the trachea. They do not cause irritation and, therefore, there are little disturbances from secretion; accordingly, they may be readily reached and removed with my bean forceps, by means of tracheoscopy under general anesthesia. All the cases known up to the present time have been rapidly and

successfully relieved (from two hours to one day after inspiration). Those treated by means of upper bronchoscopy were able to go home after the effects of the anesthesia had disappeared.

Glass Beads, Cherry-Stones, Coffee-Beans.—Noltenius, Jackson, and Pasteur observed cases in which glass beads, glass buttons, and shoe buttons were found in the deeper air passages. These bodies have a peculiar property. They continue moveable for a longer time in the trachea, and are only occasionally impacted in the right and left bronchus. They are readily coughed up, but they may be retained for a long time. Only in young children is coughing up through the larynx not so easy. This may, perhaps, be encouraged by cocainization in the subglottic space. If the bead remains, help may be quickly obtained by pushing it downwards with the applicator.

We must remember that, when performing upper bronchoscopy, the bead may be coughed up into the tube, for which reason a wide tube should be used. It is not easy to grasp a moving body in the trachea, so it is best to try to fix it in the neighborhood of the bifurcation and to grasp it there with forceps.

The same applies to cherry-stones (case of Hinsberg) and hard pills (Siebenmann). Coffee-beans (cases of Neumeyer and Fletcher Ingals) are very light and are therefore still more movable than the above, so that they may be coughed up earlier. Upper bronchoscopy is very well adapted for their removal if only a proper instrument can be used. Besides the bean forceps, a fine, sharp hook may be of service.

Prune Stones differ from the preceding by their oval shape, pointed ends, and rough surface, which are favorable for the grasp of the extraction instruments. Three cases have so far been observed by Seibenmann, Kausch and Lafite, but we may expect that sooner or later we will meet with more such cases, as the extraction of prune stones, according to reports, has been quite frequent. They occur in all ages of life. In children, they do not attain any great depth, on acount of their size. They remain in the trachea and in the neighborhood of the opening of the main bronchi. This is important to know, as the Roentgen picture is negative.

The chances for upper bronchoscopy are therefore good, as the stone lies diagonally (like coins), leaving on both sides a free space for different kinds of forceps.

Other fruit stones are occasionally found, for instance, the apricot stone in Pieniazek's case.

Pieces of Bone are the commonest foreign bodies observed through the medium of bronchoscopy. There are no less than 31 cases reported. I myself have seen six cases, Kuemmell, Spiess 1, v. Schroetter 3, Gottstein 1, Hug 1, Neumeyer 1, Just 1, Schwytzer 1, Siebenmann 1, Moure 2, Meyer 1, Kob 1, Pauns 1, Nowotny 1, Hinsberg 1, Henrici 1, Chiari 1, Garel 1, Lehr 1, Lins 1, Pauner 1, Winkler 1, von Eicken 1.

Pieces of bone are much more often inspired by adults than by children (three-fourths of the cases), and of these, mainly those above forty (half the cases). They are seldom found in the trachea, but mainly in the bronchi, especially on the right side. The trachea is more commonly the site in children.

Severe lung changes develop only occasionally directly following aspiration. On the other hand, they do not wait very long if the bone is not removed. The pieces of bone have mostly a rough surface and sharp edges and corners, become impacted and scratch the mucosa. From the small wounds, granulations spring up which enclose the piece of bone, give rise to collections of mucus and pus and destructive processes and help to shut off the dependent lung tissue. Later, putrid bronchitis, bronchiectasis, pulmonary abscess and pulmonary gangrene may appear. The secretion from the lungs under such circumstances is very abundant, making bronchoscopy and extraction very difficult. Besides this, there is a high grade of hyperesthesia of the mucosa in the neighborhood of the foreign body. It is, on this account, important to remove the inspired piece of bone as soon as possible. So far, three-fourths of the cases have been old ones.

The pieces are, as a rule, of flat shape and are 14 to 15 mm. long by 8 to 9 mm. wide. They are so situated that their long diameter corresponds to the axis of the respective bronchus and their horizontal diameter lies across the bronchus. On account of their flat form they resemble coins and are therefore easy to grasp. The granulations sometimes hide the location so that the diagonal position cannot be well made out. It is therefore necessary to clean the field with the pump in order to ascertain clearly the position of the bone. A diagonally placed bone can be easily removed with any kind of forceps.

Cubical Bodies are more difficult to remove. The bronchus may be so filled that no instrument can pass by, except perhaps a fine, sharp hook. For such cases, my assistant, Dr. Bruenings has constructed, under my direction, a sharp hook forceps which catches very well, even when only a small portion of the bone can be grasped. It worked very well in the very first case and it will doubtless be used with rapid result in many other upper bronchoscopies.

The extraction with lower bronchoscopy is naturally much easier. We make use of it when the upper is not available.

The present collection of cases does not give as yet a proper conception of the performance of bronchoscopy since there are too many technically incomplete cases included under the head.

Teeth.—Inasmuch as we have to deal with carious teeth and tooth fragments, the lung results are more severe than in the case of bones which have been sterilized by cooking. When a carious tooth gets into the lungs, severe pulmonary symptoms rapidly follow. Of 14 cases of earlier times, eight died of lung complications, one of which was from actinomycosis. Three of those who recovered went through very severe lung complications.

Such an inspired tooth, therefore, must be removed without delay. Pieniazek removed one from a patient, 40 years of age, by lower bronchoscopy, eight days after the accident. Pneumonic symptoms had already appeared in the right lower lobe, which progressed in spite of the removal, so that recovery was delayed for some time.

Pieces of Metal and Metallic Objects.—Cases of Neumeyer, Patterson, Pieniazek (3), Helfferich, J. Killian, Gottstein, H. v. Schroetter, Kuemmel, Fletcher Ingals, Hajek, 12 cases in all, including many children, especially the larger ones who like to play with such things. The special forms of such foreign bodies make the attempts at removal very difficult. Upper bronchoscopy is on this account not always successful. In one instance, the foreign body (a garter clasp) was too large and could not be drawn through the larynx. Inasmuch as the metallic bodies cause no acute lung symptoms, there is time to use upper bronchoscopy several times before the method is given up. Extraction occasionally necessitates special instruments, depending on the form of the foreign body. Bruenings' lock forceps, covered with rubber, are suggested for smooth metallic bodies.

Shirt and Collar Buttons are paricularly difficult to remove from the deeper air passages especially if the flat part lies above. If it lies diagonally in the bronchus, that is, if its neck is diagonal, it may be readily grasped and extracted, best with a hook. This is placed around the neck and the button is pressed against the end of the tube so that they may be all removed together (G. Killian, Fletcher Ingals). But when the plate lies above, it is very difficult to pass by the button. It fills the lumen of the bronchus and is covered up by the swelling of the mucous membrane. If the button remains for a longer time, granulations will spring up and, in course of time, decided stenosis may follow. In two cases of the last-named kind (Spiess, Hoffmeister), extraction was impossible. Hoffmeister's case developed pneumonia. Both cases died. I have had the good fortune to remove collar buttons three times, once with upper bronchoscopy, twice with lower. In the last two cases, the requirements for extraction were too great for upper bronchoscopy. In each case, the plate lay above and a great amount of secretion prevented its removal. The first patient, an eighteen-year-old boy, suffered from extensive bronchiectasis; he died from this condition six months later. The other patient was a boy ten years of age. The button, which was made of porcelain, caused severe pulmonary symptoms and indicated immediate extraction. Much time could not be spent with upper bronchoscopy. Lister's hook was in both cases the only instrument which I could use. It was possible to pass by the plate and grasp the bottom at the neck and thus extract it.

One of my students, Cuthbert Morton, has lately devised a spear-hook by which the plate may be grasped at its edge.

Artificial Dentures, as a rule, are too large to enter the air passages, but if they do, the larynx holds them back. They seldom pass entirely into the deep portion, and then into the left bronchus as in Wild's case. This was a plate with two teeth. It was observed eleven days after the accident by means of upper bronchoscopy and as it was too large for certain removal through the larynx, it was extracted by forceps, by means of lower bronchoscopy.

Hollow Bodies.—Fragments of canulae (Pieniazek, 3 cases; Coolidge, Neumeyer, Narath, each had one case). Hollow bodies of moderate size, pencils and the like (Thost, Neumeyer, Englemann,

G. Killian). Small hollow bodies, tin whistles, etc. (G. Killian, Claoue, Guisez). Sundries (Guisez, Durewitz, Kob).

After tracheotomy, the canula sometimes breaks off from the plate and falls down into the trachea or even into the right bronchus, seldom into the left. There is difficulty in extracting the canula by forceps and lower tracheoscopy and bringing it through the tracheotomy wound. It is best to increase this wound in size. Hooks may be used instead of forceps, if it is the outer tube with an opening in it. The hook is inserted in the opening. In one case, under general anesthesia, respiration ceased, the foreign body having completely shut off the air. The operator (Pieniazek) had the presence of mind and wisdom, in this critical situation, to push the canula down into the right bronchus, by which breathing became at once free. Later, the extraction was easy. Penholders, pencil cases and such hollow bodies, particularly if they are small as in my case of tin whistle, may cause decided difficulty, especially as they occur in children from 4 to 8 years of age. They are aspired into the bronchi and may be easily seen in Roentgen pictures. In asmuch as they are smooth metallic bodies, they occasion slight symptoms and do not cause any severe infection of the lungs. The symptoms are naturally milder when the air can pass through the tube.

The foreign bodies may well be reached by upper bronchoscopy, as happened in four out of six cases (G. Killian 2, Neumeyer, Guisez). They may be grasped at the edge with forceps whereby one branch is pushed into the opening and the other between the wall of the foreign body and that of the bronchus. If the edge of the foreign body is covered with swollen bronchial mucosa, it will be better to use my hollow body forceps, which, after being pushed in the opening, spread their branches in the hollow body and give a reliable hold. I have an instrument for narrow hollow bodies that is constructed as fine as a tipped sound. When the tip of the sound is pulled back, the four arms spread out.

II. FOREIGN BODIES WHICH ARE NOT HARD.

There is an intermediate form, with a consistency of wood and cork. I include nut-shells in this class, altogether 9 cases (Voltilini, Schmidt, Neumeyer, Pieniazek 2, d'Astros, Kausch, Cavaillon, Munasse).

As far as the technic is concerned, upper and lower tracheobronchoscopy may be used to remove the foreign body. Extraction

forceps are satisfactory. Those that break easily, such as nut kernels, must be removed with great care. (Cases of Chiari, Hinsberg, Kob, von Eicken, Nishiama.) If they are broken with the forceps, a number of foreign bodies are made out of the one, a danger whose effect will be lessened by the fact that the smaller portion may be more easily coughed up. Still one must not depend too much on this, as complication may result, especially in small children. Von Eicken's case is noteworthy. In this, a child of 14 months, a high grade of interstitial emphysema developed very rapidly, after he was unable to find the foreign body by upper bronchoscopy. Tracheotomy did not improve the emphysema, and the child died during the night. In order to prevent the breaking of the kernels, it is best to enter carefully with opened bean forceps and to grasp the whole kernel at once, and extract it at once without pressure.

Seeds of Melons, Oranges, Dates, etc.—(Yankauer, Paunz, Panner, Guisez, Saint Germain, Thomas Clayter). Our new sharp hook forceps which I have recommended for pieces of bone is just right for such fruit seeds, for they always clutch the foreign body tight.

There is a case of H. v. Schroetter to be mentioned in which pepper-corns had been inspired. These were very irritating to the bronchial mucosa and called for immediate removal. Schroetter's case, a child 11 months old, died one day later in spite of tracheotomy and lower bronchoscopy. It was impossible to locate the two pepper-corns, one of which was found on autopsy in the bronchus of the right lower lobe and one in the mouth of the bronchus of the right middle lobe.

Many cases are reported involving soft and coherent bodies, such as cotton, pieces of meat, quill of an arrow, down. (E. Mayer, Neumeyer, Hirschland, Bodmer, G. Killian.) There are, however, soft but not coherent objects, as pieces of fruit, pieces of turnip, plant leaves, etc. (G. Killian, Gottstein, Nowotny, Jackson). These things may be serious for the moment, if they are of large size; otherwise they are coughed up in parts or in toto. The extraction with forceps offers no difficulty for coherent bodies; such as are not coherent must be removed with bean forceps.

Cereal Spikes. (Cases of Pieniazek and Neumeyer). I have collected twenty-six cases in the pre-bronchoscopic period. A consideration of these cases teaches that cereal spikes are foreign

bodies which are of a very individual type. They do not remain in the position in the trachea or bronchus in which they were placed by inspiration, but gradually wander deeper, so that it is always less and less easy to seize them. They can, on this account, seldom be removed. They cause pulmonary complications and may finally be thrown out peripherally with the pus, if they are not already coughed up whole or in part. One-fourth of the patients died.

The two cases observed under bronchoscopy were not much better on that account. One, a child of eight months, died in spite of the extraction: in the other, the object was not seen and the expulsion was not noted.

We should treat these cases as soon as possible. In making the extraction, it is to be understood that the spikes have their points directly upwards—i. e., against the line of extraction. They can be completely removed only if they are completely surrounded by a properly fenestrated forceps.

Expansible Fruit-kernels, Beans, Etc. Bean cases (Pieniazek, 3; G. Killian, 2; Nahrkorn, 2; Kuemmel, Harrington, von Eicken, Gottstein, Siebenmann, Kausch, 2; E. Mayer, New York, Nowotny, Pommering, Kahler; eighteen cases in all). In addition, three similar cases (H. von Schroetter, Paunz, Fletcher Ingals). Beans are the most commonly inspired foreign bodies. Over 100 cases were reported in pre-bronchoscopic ties. The number under bronchoscopic examinations reaches eighteen cases. We must, therefore, be assured that we shall have much more to do with these serious foreign bodies, involving small children, generally.

Beans are at the beginning movable, but become fixed as soon as they swell, in the opening of the bronchus, much more frequently in the right than in the left. By swelling, they shut off the dependent lung tissue and as a rule the whole lobe. Bronchitic secretion collects below them and pneumonia symptoms with high fever and extensive atalectasis soon develop. Thirty-nine per cent of former cases die.

As these complications occur very soon, indeed, in the course of a few days, we cannot operate too promptly upon such a case. In addition we meet with the very unpleasant changes caused by the swelling, which makes the bean soft and crumbling.

So far there has been little satisfaction with upper bronchoscopy. In only two cases was it in some measure successful, and once

suffocation followed because one-half of the bean fell into the sound lung and shut it off. Secondary tracheotomy was required in two other cases.

Apparently the subglottic portion was too severely irritated by the frequent passage of the tube for removal of the fragments.

Lower bronchoscopy, which I must most earnestly recommend, was uniformly successful in thirteen cases—that is, the bean was removed. However, two died suddenly and others suffered for a long time from broncho-pneumonia before they were relieved. I still cling to the hope that we shall secure better results by earlier treatment, improvements in instruments and additional experience.

I have had constructed for the extraction of beans a special forceps which lately has been improved by my assistant, Bruening. It surrounds the bean after the fashion of an obstetric forceps, spreading easily by pushing (in order to dilate the bronchus and damage the swollen bean as little as possible) and closing with delicate pressure. Practice will demonstrate whether this instrument will meet all indications.

RESUME OF THE RESULTS FROM TRACHEO-BRONCHOSCOPY SO FAR ATTAINED.

If we endeavor to give an account of what has been attained by the new method of treatment of foreign bodies, we must consider that the 164 cases, published up to the present time, show many imperfections and comprise a time which can be called the infancy of tracheo-bronchoscopy. In some instances the upper method was not undertaken or failed, nothing could be seen, the extraction could not be carried out, or practice, experience or necessary instruments were wanting—introducing tube, exhaust pump and especially extraction instrument which had not been anticipated. The same things have played a great role in the easier lower bronchoscopy, so that many things in unexpected ways caused the operator to work in the dark after he had seen the foreign body.

In spite of all of this, most remarkable results have been secured. Most of the patients were relieved by one or the other method, and sometimes nature herself assisted by causing the foreign body to be coughed up (eight times). But of 159 cases (leaving out five with unknown result), twenty-one—13 per cent—died, for which the new method is but slightly responsible—two deaths from cocain, two in which it was impossible to remove the object on account of bronchial stenosis and one from suffocation, in spite of

upper and lower bronchoscopy. The remaining sixteen died of pulmonary complications, five with the foreign body in the lung, and the others in spite of removal. Even a casual glance shows that early extraction will reduce the mortality to a minimum.

Upper bronchoscopy has been fully successful in fifty-four cases and lower bronchoscopy in sixty-three.

My own statistics give perhaps a better judgment for the future of cases of foreign bodies in the deeper air passages than the general, since I have gradually acquired a larger experience and practice. Nevertheless, I have the impression that in many cases my technic has not reached the highest mark and I hope to obtain better results in the future. As shown by the list of cases, only one death resulted in the eighteen cases and this was six months after the removal of the foreign body, caused by severe lung complication, due to its long sojourn in the air passages. In only two cases was I unable to find the foreign body and in only one was I unable to remove it on account of its being coughed up.

Upper bronchoscopy was performed in twelve cases, upper and lower in five and lower tracheo-bronchoscopy alone in one. However, I hope in the future, with improved technic, to be successful with the upper method at the first sitting and to use the lower only in the severest cases.

May the future bring this operation into general use.*

MY FOREIGN BODY CASES TREATED BY TRACHEO-BRONCHOSCOPY.

Needles:

Case I. I was called on in 1900 to treat a young man who, ten years before, when a school boy of 14 years, inspired a long needle with a black glass head, while playing with a pea shooter. The immediate symptoms were cough and attacks of dyspnea, which gradually disappeared. Two months before, following bodily exertion, there was a slight pulmonary hemorrhage, which did not recur.

The needle was visible in the Roentgen picture, in spite of its ten years' sojourn in the lungs. I made three upper bronchoscopic examinations of the right lung without finding the foreign body. Then I made lower bronchoscopy twice, after tracheotomy. The main passageway and the orifices of the lateral passages of

* Since I wrote this, I have seen the foreign body removed by upper bronchoscopy at the first sitting, twice in my clinic and twice on my American trip. The first was by Dr. Von Eicken, the second by Dr. Bruenings, the third and fourth by me in New York and Chicago respectively.

the right bronchial system were carefully examined without finding the needle or any evidence of its presence. I surmise that it had stuck into the branches of the middle lobe or at least into the tissues in this neighborhood. Patient has remained well.

Case II. A druggist who for fifteen years was addicted to morphin had inspired the needle of a Pravaz syringe. The symptoms, which were mild at first, soon developed into a sticking pain in the chest and irritative cough. On the sixth day, the needle was observed by X-rays in the right lung, where it lay quite deep. Its shadow corresponded to the fourth rib anteriorly and to the seventh or eighth posteriorly. The head of the needle was below. When the patient came to me on the ninth day for examination I found numerous moderate ronchi over the right lower lung, especially on inspiration, but no dullness. Two days later, eleven after the accident, I undertook upper bronchoscopy under general anesthesia, after the patient had received an injection of his maximum morphin dose (0.06 gram.). The needle was discovered 34 cm. from the upper teeth, by means of the tube 35 cm. long and 11 cm. in diameter. I removed the needle, which was inclined from below and outwards to above and within; its point was inserted in the medial bronchial wall and covered with a granulation. The needle lay substantially in the main bronchus of the lower lobe, and its head stuck in one of its branches. I carefully cocainized the neighborhood of the needle. The patient thereupon coughed, forcing the point of the needle free and it could then easily be grasped by von Eicken's forceps. The extraction was rapidly accomplished through the tube. The rales in the right lower lobe disappeared in a short time. Recovery.

Pebbles:

Case III. Boy four years old, August, 1904. An hour before, while playing, he had aspired a foreign body of unknown character. Slight dyspepsia, marked tracheal stridor. Cough, slight and infrequent. Chloroform anesthesia and bronchoscopy with a tube 21x0.7 cm. The foreign body was seen near the bifurcation, sticking partially in the right main bronchus. It was of a flat shape and could be readily extracted with tube forceps. The pebble was 13 mm. long, to 9 mm. broad and 2 to 3 mm. thick.

The child slept a short time, waked up happy and pleasant and went home three hours after the extraction. Five hours had elapsed

between the inspiration of the pebble and the return of the patient home. Recovery.

Pieces of Bones:

Case IV. Man, sixty-three years old, on March 27, 1897,, while eating soup made of pork, inspired a bone. Cough, dyspnea, slight bloody expectoration. Three days later, direct upper tracheoscopy under cocain with patient in the sitting position. The tube was only 25 cm. long and 9 mm. in diameter. For all that, I was able to see the foreign body in the entrance of the right main bronchus and after some fruitless efforts I succeeded in removing it. The object was 17x14x8 mm. and was composed of spongy bone substance. Cortical bone was found in only one place. Patient left for home next day. Recovery.

Case V. Man, 42 years old, inspired a piece of bone, in March, 1896. Severe lung symptoms, pneumonic and pleuritic processes and bronchiectasis developed, until he was subjected to upper bronchoscopy on Dec. 21, 1899, under cocain and morphin. The bone was seen as a diagonally placed plate 3 cm. deep in the right main bronchus. It was very difficult to engage on account of extraordinarily abundant mucus products, but after some failures it was removed. It was 15x11x4 mm. The symptoms of the patient soon improved and after a year or so he was relatively well.

Case VI. Woman, 33 years of age, with slight tubercular infiltration of the left upper lobe, six months pregnant, inspired a piece of bone, fourteen days before. Since then, cough almost to suffocation. Upper bronchoscopy, under morphin cocain anesthesia, with a tube 35 cm. by 0.9 cm., patient in the sitting position, showed the bone in the entrance of the bronchus of the right upper lobe. Unfortunately, it was pushed with a cocain applicator into the right lower lobe and it stuck there so tightly that it could not be removed by a second upper bronchoscopy. The mucosa was swollen around it and was covered with fibrinous membrane. I performed tracheotomy and, several days later, used lower bronchoscopy with a tube 23 by 0.7 cm., the patient being in the recumbent position. In spite of the abundant mucus and pus, it was possible to pass the Lister hook beyond the bone and to effect its removal through the tube. Its dimensions were 8 by 7 by 5 mm. Immediate removal of the canula. Recovery. The signs over the left apex had practically disappeared six months later.

Case VII. A woman, 20 years of age, came under my treatment, March 14, 1903, on acount of a piece of bone which she had inspired in the middle of September, 1902. The sequelae were severe cough, shortness of breath, hemoptysis. On examination, a loud rale was heard posteriorly and inferiorly, strongest over the eighth rib on the right side. Upper bronchoscopy with a tube 24 by 0.9 cm. was performed the next day, with hanging head, under morphin chloroform anesthesia, as the patient was very nervous. The bone was found in the first lateral branch of the bronchus of the right lower lobe. In order to reach it well, it was necessary to introduce a tube 34 by 0.7 cm. through the former. The bone was situated 31 cm. from the upper teeth. The forceps was unable to engage, but the Lister hook was successful. It was supported by the end of the lower tube and the bone was drawn up, together with both tubes. In this way, unfortunately, it was lost, so it was again necessary to introduce the tube and the extraction was accomplished by Lister's hook at the second effort. The bone, which was of cortical and spongy substance, was 13 by 11 by 9 mm. in size. Recovery rapid.

Case VIII. Man, aet. 56. Inspiration of a piece of bone, February 7, 1904. As the patient suffered a great deal, he was put under morphin chloroform narcosis on May 21, 1904. Under upper bronchoscopy with a tube 32 by 1.1 cm., I found the piece of bone in the left main bronchus and, grasping it with the forceps, accomplished its extraction. The foreign body, covered with bad smelling pus, measured 2 by 1 by 0.5 cm. Recovery rapid.

Case IX. Man, aet. 23. Inspiration of piece of bone, Mar. 29, 1906. Mild pulmonary symptoms soon appeared. On May 23, under morphin chloroform narcosis, upper bronchoscopy was performed with a 9 mm. tube. It was impossible with the forceps and hook to grasp the bone, which was found in the right main bronchus, on account of the severe coughing paroxysms of the patient. The bone sank deeper so that it was not possible any more to get beyond it. I, therefore, performed tracheotomy immediately, and after some days, lower bronchoscopy with morphin cocain anesthesia. It was even still more difficult to take out the foreign body, first with fine, sharp hooks and then with the blunt Lister hook. Canula immediately removed; signs of atelectasis of the right lower lobe disappeared after a week. Size of the bone 13x13x7 mm. Recovery.

Shirt and Collar Buttons:

Case X. Man, aet. eighteen, a half year before inspired a shirt button into the left lung, followed by severe pulmonary symptoms, which developed into extensive bronchiectasis with tremendous pus production. It was unfortunately not possible to find the foreign body, although upper bronchoscopy was twice performed. I was forced, therefore, to perform tracheotomy and lower bronchoscopy on July 20, 1901, under morphin cocain. The extraction was, however, very difficult, inasmuch as the collar button had, through the previous fruitless efforts, been forced deep into the division of the left lower lobe bronchus. Finally, the button was held against the bronchoscopic tube with a Lister's hook and extracted, whereupon the pulmonary symptoms rapidly improved. Six months later, however, the patient was attacked with suppurative pleurisy on the other side, and died.

Case XI. Man, aged 40, inspired a shirt button on Dec. 27, 1902. Two days later, upper bronchoscopy was performed under cocain with the patient in the sitting position. The button was found deep in the left lower lobe bronchus. As it was placed diagonally, it was easily grasped with the hook, held against the tube and removed. Recovery.

Case XII. Boy, aged 10, inspired a porcelain collar button on August 20, 1905, followed by severe pulmonary symptoms with high fever. Upper bronchoscopy on the evening of the third day, under chloroform anesthesia, with a tube 9 mm. in diameter, which was introduced through a tube spatula. The button stuck in the left main bronchus with its plate above. No instrument, not even the hook, could pass it, as there was no space between the plate and the bronchial wall. Tracheotomy. Two days later, lower bronchoscopy. Extraction was accomplished with the Lister hook only after numerous fruitless efforts, as the head was so smooth that it always glided off. The lung symptoms disappeared after a few days. Early recovery.

Small Hollow Brass Bodies:

Case XIII. Boy, aged 8, twelve weeks ago (1903) inspired a brass case into the right lung. Upper direct bronchoscopy, with hanging head, under general anesthesia, with a tube 7 mm. in diameter, which was introduced through a tube spatula. The case was engaged in the lower end of the tube and was removed without any other agency. Recovery.

Tin Whistles:

Case XIV. A girl of seven, inspired a small tin whistle belonging to a playing instrument, on April 18, 1904. Very severe paroxysms of coughing immediately appeared and an inspiratory stridor was heard. The other usual bad symptoms were absent. The Roentgen picture showed the foreign body at the level of the seventh rib behind on the right side. It was necessary to perform upper bronchoscopy three times under general anesthesia before extraction was satisfactorily accomplished, August 4, 1904. A tube 20 by 0.7 cm., was used, through which a second 5 mm. in diameter was introduced 5 cm. farther down. For extraction I had a special instrument made with four arms, which spread out when the head was pulled back. This was introduced into the hollow portion and thus the extraction was accomplished. Two days later the lungs were entirely free. Recovery.

Down Feathers:

Case XV. Woman, aged 42, who had for twenty years been engaged in picking chicken feathers, had been attacked with diffuse bronchitis for three years. She had occasionally coughed up down feathers. As it was possible that there were other feathers in the lungs, I performed upper bronchoscopy in January, 1902, under cocain, with the patient in the recumbent position. It was not possible, in spite of the most careful search of the bronchial tree, to find a single feather, yet she coughed up such a one a few days later.

Case XVI. A nine year old boy inspired, August, 1898, a piece of pear into the right lung, which could be seen January 3, 1899, by means of upper direct bronchoscopy, under general anesthesia. It lay at the opening of the bronchus of the right middle lobe, and was covered in part with granulations. Inasmuch as a proper extraction instrument was lacking, the removal of the foreign body was postponed. Four days later, bronchoscopic examination showed that it was not present, since it had been coughed up in the meantime. Recovery.

Beans:

Case XVII. Boy, four years old, had inspired a large bean on the morning of September 27, 1898. At the beginning there were slight disturbances. In the evening, however, dyspnea and cyanosis appeared. The right half of the thorax was fixed on breathing. On the following day I performed tracheotomy on account of the

dyspnea, whereupon the bean, which was still movable, appeared, but was immediately inspired into the depths and could be removed only with a sharp hook under lower bronchoscopy. In the beginning, it had obstructed the right main bronchus. It was 10 by 18 mm. in size. The child recovered in a few days, after slight bronchitis.

Case XVIII. Boy, aged 6, inspired a bean on Oct. 31, 1900. On the evening of November 3, the right lower lobe was markedly dull, and the temperature 39.1 C (102.4 F.). Upper bronchoscopy, under general anesthesia, showed a swollen bean in the right main bronchus. It was impossible to engage it with the sharp hook. It was possible to reach it with the dull hook and to draw it into the trachea. A quantity of purulent mucus came out of the right bronchial tree. A piece of the bean cortex was coughed out; the remainder lay movable in the trachea, striking the cords violently on expiration. When I reintroduced the tube rapidly, the breathing was disturbed, as the bean was pressed against the tube on expiration. I pushed it back with forceps and engaged it, which caused it to break into pieces. Another piece of the cortex, and some of the smaller fragments, were coughed up; others I removed with forceps and hook through several introductions. One-fourth of the bean was still lacking, which must have been coughed up, as the most persistent search of the right and left bronchial system failed to reveal it. The temperature rapidly sank, the child improved, and ten days later had recovered from his bronchitis, and was discharged.

ESOPHAGUS.

My clinic provides, in the course of the year, a rich material in foreign bodies in the esophagus.

We performed esophagoscopy in all cases where the patient thought he had swallowed a foreign body, and we consider this method most satisfactory from a diagnostic and therapeutic standpoint.

The character of the foreign bodies which occur in the esophagus is not so varied as in the respiratory tract. They are mainly pieces of bone, meat fragments, artificial dentures, or parts of the same, and coins, especially in children. Needles, fishbones and fruit seeds (plum), are occasionally seen. All other things are uncommon. Pieces of bone, meat fragments and dentures may be quite large. Small pieces of bone and food come into play, especially when there

is stenosis of the esophagus. They may be impacted above or within it.

All foreign bodies in the normal esophagus are caught in the neighborhood of the cricoid, at the beginning of the esophagus, which is kept closed by muscular action and is opened only on deglutition. Everything that is not soft, smooth, and readily passable, and exceeds a definite volume, is lightly held in this place. In all cases in which a bougie has not been used on the patient, we may be certain that the foreign body is at the beginning of the esophagus near the lower portion of the larynx. Needles, fishbones, and nails usually stick in this place and glide down slowly with the food into the deeper portions. As a stenosis may easily result on account of the impaction of the foreign body in the entrance of the esophagus, the saliva itself is difficult to swallow. This makes the examination of the sitting patient very unpleasant and uncomfortable for physician and patient, on account of the excessive salivation. I prefer, therefore, to have such patients lying down on the right side, or the left side, whether under general anesthesia or not. The operating table must be so arranged that the shoulder and head lie somewhat lower, so that the saliva, which is collected, runs out of the corner of the mouth.

Cocainization is employed while the patient is in the sitting position, and general anesthesia is used only in children. Care must be taken not to push the foreign body deeper during cocainization. It passes easily into the stomach, and we may search in vain for it. This is no great pity, as experience shows that, with favorable diet, the foreign body passes off in the usual way.

Sharp bodies, needles and nails, however, may occasionally cause annoyance. One of my patients could very easily feel the pin in his stomach, and in the evening in the cecal region. Another patient had very severe pains in the abdomen, as long as the needle journeyed through the intestines. Once we feared complications; however, everything went well.

When the foreign body slips into the stomach, the patient has the distressing feeling, until it passes away, that it may still cause unpleasant symptoms. It is, therefore, desirable for him, and for the esophagoscopist as well, that the corpus delicti be removed and exhibited.

Esophagoscopy is performed with a tube spatula which is introduced without insertion of the finger, under good light from a

hand mirror or forehead lamp. The patient holds his tongue, in the beginning. The collected secretion is removed with the exhaust pump. We approach the foreign body slowly with the tube spatula and locate carefully for the purpose of extraction.

Coins, pieces of bone, artificial dentures are situated in a flat position in the esophagus, in a frontal direction. We introduce a properly formed clutch forceps or bean forceps with branches in front and behind the foreign body, and then it may be easily removed. The highest portion should be engaged, so that it may not attach itself to anything and make it difficult to remove. When a sharp point has been caught, it must be pushed backward. Most of the bodies are too large to pass through the tube, and so are removed with it. There is an advantage, in that the tube dilates the way for the foreign body.

The resistance to be overcome in extraction is stronger when the mucous membrane over the foreign body is swollen by inflammation. No force should be used in overcoming this resistance. Impacted bodies may often be drawn some distance upwards, since the esophagus may be stretched. There comes a time, therefore, when the resistance is so increased that there is danger of tearing the esophagus. This condition is recognized when the released foreign body flies back immediately into its old location.

Coins are most certain of extraction, since they are smooth and are unable to wound anything. They can be drawn out with force, without the likelihood of doing harm. Coins should not be removed except by means of the esophagoscope. I do not consider that esophagotomy in this respect is a modern procedure any more. We must energetically defend this proposition. Not less should we oppose the use of coin removers. These cases are always in children, and it is just in such that the coin removers usually purchased may do harm. I once saw a case in which a colleague had slit almost the whole esophagus with a coin remover, after which he was unable to remove the instrument. After it was removed at the surgical clinic, I observed the wound by means of the esophagoscope. Of course the child soon died from suppurative mediastinitis.

Fruit seeds are to be treated like coins. Fishbones or needles may be grasped with von Eicken's forceps in the neighborhood of the point and removed.

Remains of food and pieces of meat slip very easily into the stomach if some space is made by cocainization or by pressure with the tube. If they remain, they must have a very considerable caliber. Then the extraction may be difficult and of long duration. The forceps tears away the portion grasped, and so only a piece is removed. On this account, it is necessary often to re-enter, unless a very broad portion is seized. On the other hand, if a broad portion of the piece of meat is grasped, it may happen that on account of its size it finds so great resistance behind the larynx that it is impossible for it to pass, as happened in one of my cases. Only after I had gradually reduced it in size was the extraction successful.

Pieces of bone are not always of satisfactory form. Old persons who have impaired sensations of taste in the mouth, on account of a defective set of teeth, may swallow large bones unconsciously, without having a clear understanding of their size or form. It thus happens that there are really serious bones with sharp lateral processes which cannot be seen by esophagoscopy. These may become impacted upon extraction. If these do not glide readily, great care must be taken in extraction and success accomplished by pushing backward, moving to and fro, and grasping in different places. If not successful without force, esophagotomy is far better for the patient.

What has been said is true of artificial dentures. Here at least we have the advantage of knowing, from the patient, the size and shape of the foreign body swallowed. On this account, we are able to look out for projections and hooks. Besides, we will know whether or not the plate is made out of rubber or metal. In spite of all difficulties, many artificial plates have been successfully removed. I warn, however, the very energetic, against the exhibition of force. If there is a wound of the esophagus, severe symptoms will rapidly occur, which can be met only with early mediastinotomy or the patient is surely certain of death. If the extraction, by the natural way, appears risky, then esophagotomy is to be preferred. It is only when the artificial plate is not made out of metal and contain no metal that it will be possible to cut it up into pieces by the galvano-cautery snare, which was satisfactory in one of my cases. The snare is made narrow, and is pushed far beyond the plate. The wire is then pushed to one side (since the snare is unfolded better in this way). There is now a large snare

wire beyond the plate which catches it when drawn upward; it can be easily cut by a very small amount of heat. Perhaps there will be constructed harmless instruments which may cut metallic artificial plates within the esophagus.

I have been assuming, up to this time, that the foreign body is in the upper portion of the esophagus. It may, however, as has already been stated, be pushed deeper and deeper by the tube spatula and during the efforts at extraction, even as far as the cardia. The practitioners into whose hands the case first comes are often responsible for pushing it down. If otherwise there is no success, they exert themselves to push the foreign body into the stomach. We then usually find severer symptoms.

A simple tube spatula is no longer sufficient, so it must be lengthened by the introduction of tubes which will pass directly to the foreign body.

Naturally, the different depths required an extracting instrument which corresponds in length. The distance to the cardia in tall men is 50 cm. from the upper teeth. Accordingly, we must be prepared for this condition. The extraction is made more difficult on account of working in the deeper portions, but it is not so much as the uninitiated think. One may soon learn to throw a good light through the long tube sufficient to see and to carry on the technic of extraction. Even complicated treatment may be undertaken in the neighborhood of the cardia, as shown by the case reported of cutting up of artificial plate within the esophagus.

It is much more serious when a larger foreign body, which cannot be cut up, is impacted in the deeper portions. In such cases gastrotomy must be performed and the foreign body removed from below. Posterior mediastinotomy was in one case successful, but, unfortunately, this operation is still so dangerous that it is almost always followed by death.

If the foreign body lies above or within a stenosis, the same rules apply for examination and extraction. One will hardly meet with greater difficulties; under certain circumstances the patient may be saved from a gastrotomy already planned.

There are cases also in which esophagoscopy is contraindicated from the beginning. It is best to refrain entirely if the symptoms indicate that there is a perforation of the wall of the esophagus; i. e., intense pain in the neighborhood of the perforation, increased temperature, emphysema of the skin. We should look out for such

symptoms, especially if some one else has dealt with the patient. The esophagus may cut itself on the sharp surface if a strong effort at swallowing is made.

A perforation necessitates an external operation. The tissues in the neighborhood are very rapidly infected and must be very widely opened and the point of perforation found. It is therefore best to leave the foreign body alone and to make an esophagotomy; in this way the position of the perforation will be more easily found. As to the question whether to open on the right or the left side, the greater pain decides. The chances for cure are better in the high perforation. We were successful in two patients. A patient with deep perforation died in spite of mediastinotomy from the neck. Here deep posterior mediastinotomy bespeaks better results.

I must still mention that, instead of a foreign body, there may be only scratches, especially in the neighborhood of the pharynx and entrance of the esophagus, which make the patient believe that the foreign body still sticks there unless we demonstrate the opposite by esophagoscopic examination. I do not believe that a foreign body in the esophagus can be overlooked under proper technic.

If I exclude the two cases in which the patients already had severe wounds of the esophagus, I have no deaths to report; all were promptly cured. This is certainly a good result in 17 cases.

FOREIGN BODIES IN THE ESOPHAGUS. CASES FROM THE CLINIC TREATED BY G. KILLIAN AND C. VON EIKEN.

Case I. Man, aged 62. September 24, 1898. Two hours before, he swallowed a bone, since which time he has had severe pains in the neighborhood of the cricoid cartilage. Difficulty in swallowing. Esophagoscopy. By pushing the tube behind the cricoid, the piece of bone was observed for a moment; it was freed from its lodgment and, with lightning rapidity, disappeared in the depths. After several days it passed off in the natural way. It was of spongy material, 1 cm. long by 0.75 cm. wide.

Case II. Woman, aged 26 years, March 7, 1903. Yesterday evening, patient swallowed a small chicken bone. Severe pain below the larynx. With the laryngoscope, a blood blister was seen deep in the left sinus pyriformis. Esophagoscopy with a tube spatula 14 by 1.3 cm. A small and pointed bone was found in the upper portion of the esophagus impacted in the mucosa; it was

grasped with a forceps, loosened and removed; 12 by 2 mm. in size. Recovery.

Case III. Woman, aged 79. Patient swallowed a bone while eating soup two days ago. The physician made fruitless efforts to remove the foreign body. Severe pains appeared the following night, which kept the patient from swallowing anything. An examination for extraction was made on the third day after; we found hemorrhage on both sides of the palate and in the left sinus pyriformis. The left ventricular band and vocal band were decidedly reddened. Esophagoscopy. At first a short tube was used and then a tube 39 cm. long by 9 mm. in diameter. Just above the cardia a large piece of bone projected with a sharp edge upward. Extraction with the tube. Recovery.

Case IV. Man, aged 59 years, on December 29, 1901, swallowed, with his soup, a piece of bone which remained in the neighborhood of the cricoid. Patient ate bread afterwards and thereby the bone passed down somewhat deeper. A physician sought to push the foreign body into the depths with a sponge probang, but was succesful only for a short distance. Attempts with other instruments were without success, and the instruments were covered with blood when they were removed from the esophagus. Patient had no more pain afterwards. At about nine o'clock of the evening of the same day, under esophagoscopy, with a tube 14 mm. in diameter by 25 cm., measured from the upper teeth, the esophagus was found filled with blood clots, among which shreds of tissue were visible. After thoroughly cleaning the field of view with the exhaust pump, a very severe wound of the esophagus was observed. At the same time a brownish red body presented itself, which was found on extraction, to be a piece of meat. Another introduction of the tube led to the finding of the bone. Its point was grasped by the forceps. There was enormous resistance upon drawing it, whereupon the foreign body was pushed some distance deeper by the tube and then removed. An unusually large, flat bone came to view, 3.6 by 2.8 cm. large, which on one side presented long prongs which were arranged opposite the apex of the bone and which acted as barbs. At the beginning, the patient's symptoms were moderate, the pain lessened, but later there was additional pain on the right side. Morphin injection. Temperature the next morning 38.4 C. (101.1 F.). External mediastinotomy was performed in the surgical clinic by Kraske. The esophagus was

stretched and infiltrated, and was followed 10 cm. into the chest. Wide opening of the tissues, but perforation not found. Increased symptoms and fever. Death in one week. Autopsy showed a large tear which began 10 cm. below the cricoid and was 5 cm. long. This led into a putrid abscess in the right mediastinum, opposite of the hilus of the right lung. We had been unable to find the tear, because we were operating on the wrong side. This would have been prevented if an esophagoscopic examination had followed the extraction. This was attempted but the electric light failed.

Case V. Man, aged 20, swallowed a bone of a hog on August 12, 1905, which was pushed down by the physician in attendance. Pain was experienced 35 cm. from the upper teeth. Fluids only could be swallowed. Esophagoscopy was performed two days later with the patient lying on the left side, the tube being 42 by 1.4 cm. The bone appeared at a distance of 36 cm. in a diagonal position. It could be easily grasped but it slipped away at 20 cm. and was again grasped, after which the extraction was successful. Secondary examination showed a small wound of the mucosa where the foreign body lay. Recovery.

Case VI. Man, aged 21, had swallowed, on that morning, a piece of chicken bone which stuck 22 cm. under the cricoid. Esophagoscopy, with a tube 30 by 0.7 cm., revealed the bone, surrounded by remains of food. Fruitless efforts with the bean forceps were followed by success with the toothed forceps. Length of bone, 3 cm. Course good.

Case VII. Woman, aged 69, had, since day before yesterday, that is November 13, 1906, a bone in her esophagus. Pain behind the cricoid. The attending physician made four attempts to push it down. Severe pain followed in the lowest dorsal vertebra. Temperature 37.5 C. (99.5 F.) Laryngoscopy. The bougie discovered a resistance at 38 cm. Esophagoscopy, in the right lateral position, with a tube 42 by 1.1 cm. At a depth of 36 cm., a sharp horizontally placed piece of bone was observed. This was grasped with the forceps, but was caught in the mucous membrane with its upper end, upon being drawn out, so that it became necessary to release it. Upon new efforts being made with the tube, it suddenly disappeared into the stomach. Course good.

Case VIII. Man, aged 43, while eating noodle soup, swallowed, at 11 o'clock today, a bone which remained sticking in his throat. The patient ate a great deal afterwards, in order to force the

bone down, but the attempt was not successful. Severe pains in the jugulum. Esophagoscopy with tube spatula three hours later. Only after removal of some pieces of meat could the bone be seen and extracted with the toothed forceps, after overcoming a certain resistance. It was as thick as a small thumb with an apex upward and a broad portion downward. Some scratches were discovered by subsequent examination. Patient soon left for home. Recovery.

Case IX. A boy, aged 1½, swallowed a coin on January 13, 1903. Several hours later this was found by means of Roentgen rays, sticking under the cricoid where it could be felt with the finger. Slight general anesthesia. Tube spatula. Extraction with forceps. The coin was a ten pfennig piece.

Case X. Girl, aged 9, in August, 1904, two days before swallowed a coin, which was visible with the Röntgen rays under the cricoid. Esophagoscopy, patient on the left side. A tube 18 by 0.7 cm. was introduced with a guide tube. The edge of the coin came into view under an inflamed swollen portion of the mucous membrane. The inflammatory swelling caused a distinct resistance to extraction. Recovery. The coin was a two kreutzer piece of 1815.

Case XI. Girl, aged 10, had swallowed a two pfennig piece two hours before (August 10, 1906). Difficulty of swallowing. Coin visible in Roentgen picture under the cricoid in a frontal position. Esophagoscopy in sitting position under cocain, with a tube spatula, 12 by 0.7 cm. The coin could be seen but could not be grasped on account of the great salivation. Thereupon esophagoscopy was performed in the lateral position with a longer tube. Saliva was drawn off with the exhaust pump. The coin in the meantime slipped into the stomach and passed off in a few days with the feces. Recovery.

Case XII. Child of 2½ years, had swallowed a five pfennig piece twelve days before October 10, 1906. Transitory fever. Swallowing of food difficult, milk only being possible. The child had suffered for some time from an extensive infantile paralysis. The coin was visible in the Roentgen picture in the upper portion of the esophagus. Esophagoscopy, under general anesthesia, on the right side. Introduction of a tube, 5 mm. in diameter, could only be accomplished by bending the head as far back as possible. A resistance was encountered in the upper portion of the esophagus, which appeared as a circular stenosis with whitish deposit on

the mucous membrane. After cleaning the field with the exhaust pump, the upper portion of the coin was visible. The esophagus opened up at each inspiration, so that it was easy to grasp the coin at that moment. There was some resistance in the neighborhood of the stenosis. Recovery from the effects of the foreign body.

Case XIII. Man, aged 67, had swallowed, on the evening of the day when he came to me, December 29, 1906, a large piece of solid meat of a hog's heart, without much chewing. Patient was in a hurry; he had no teeth. The piece of meat remained sticking in the esophagus. Upon breathing, rales were heard which could be clearly established by the mirror, as caused by the collection of saliva over the larynx. He could not swallow the saliva, as the esophagus was completely obstructed. He could clearly feel that the piece of meat lay high up at the orifice of the esophagus. Esophagoscopy in the left lateral position, under cocain. The mass was grasped by aid of the tube spatula, but upon extraction only a piece was torn off. This was repeated several times. As a result, the piece of meat descended, so that a longer tube was required. The extraction was again not completely successful, only the portion grasped being removed. It was then possible to draw the mass again as far as the cricoid. After a short pause, the tube spatula was again introduced. The mass again descended, so that it could now be reached only with a 40 cm. tube; still I was able to raise it after removal of a piece, but it once more slipped down. In the endeavor to grasp it in this place, a small piece of the mucosa was caught, causing it to bleed. I made an attempt to push the mass into the stomach, but was unsuccessful, as it was too large. There was nothing left except again to perform esophagoscopy, with a tube 50 cm. long, using one of my longest toothed forceps, about 60 cm. long. I soon grasped the mass of meat and drew it up behind the cricoid, where I could bring the tube spatula into action. The forceps tore off a large piece. The mass descended slightly, so that a longer tube was again necessary. It had become so much smaller in volume that it could finally pass by the cricoid. The whole procedure lasted about an hour and a half. In spite of it all, the patient was comparatively little affected. I would not permit him to eat until the second day following, on account of the wounding of the mucous membrane; he was only given some alcohol from time to time. He had some

little pain in the throat and slight elevation of temperature. However, the symptoms disappeared by the third day and the patient could soon be discharged as cured.

Case XIV. Woman, aged thirty-seven, on March 3, 1907, while eating soup, swallowed a small plate with two teeth, and came immediately to the clinic. The plate could be felt externally under the cricoid, and it was shown in this place by the Röntgen picture. Esophagoscopy with the tube spatula 20 by 1.2 cm. The plate came immediately into view at the opening of the esophagus and was easily removed. Recovery.

Case XV. A woman, aged 32, had swallowed a tooth plate. The Röntgen rays examination gave no result, but with the probe a foreign body could be felt in the esophagus. Attempts to remove or dislodge it were unsuccessful. I saw the woman for the first time eight weeks after the accident. She had, in the meantime, lost strength considerably, not being able to take sufficient nourishment. An examination with the esophagoscope was made and I found the foreign body impacted in the esophagus at a distance of 35 cm. from the upper incisors, therefore near the cardia. I attempted to extract the plate by means of a tubular forceps, but as the resistance offered was so great that I was afraid of injuring the esophagus wall, I had to give up further attempts in this direction. I now began to consider the possibility of diminishing the size of the foreign body and of removing it piecemeal. At first I was able to burn off only a small part, but at a third attempt I succeeded in cutting the plate into two parts by means of a galvanic snare. I introduced this snare with a small loop only, at the side of the plate; then I gradually enlarged the loop by pushing the wire forward on one side only, and then drawing it back. I caught the foreign body, and was able to cut it through with incandescent wire. The two pieces were easily removed with forceps. I take pleasure in showing them to you here. The patient recovered without interruption. (*British Med. Journal,* August 30, 1902.)

It should be reported that we have, during the past years, examined 17 cases by means of esophagoscopy, in which the foreign body was not present at the time of the examination; scratches only could be found in a portion of the cases. All remained without any bad results. In some, the foreign body soon passed away; in others, it is doubtful if there had been one present. In the last

case of this sort, I found a small beginning carcinoma instead of a foreign body. The two following cases may be of interest, although esophagoscopy was not undertaken; it was contraindicated.

Case XVI. A young man came, in the summer of 1904, to our clinic early in the morning, after he had swallowed a fragment of glass on the evening before. It was very hot, and he was very thirsty; he had, with avidity, taken a glass of beer. Although he had broken the glass on the edge of the table, he nevertheless drank it very rapidly, as a result of which a piece of glass stuck in his throat. This could be seen by the Röntgen picture behind the larynx, five or six hours later, when the patient came under our observation. He was suffering greatly and had continual paroxysms of swallowing accompanied by severe pain in the throat. The temperature was 37.8 C. (100 F.). Examining his neck, I found an emphysema of the skin, establishing the existence of a perforation of the esophagus. Evidently the sharp edge of the glass had cut the mucosa and the constrictor. Bloody mucus was seen behind the larynx by means of the laryngoscope. Immediate esophagotomy was indicated in this case. This was performed in our surgical clinic by Dr. Pertz, on the left side, where the pain was greatest. The perforation was found here on the lateral wall of the hypopharynx; after dilatation, it was easy to remove the foreign body. Patient recovered after suffering for some time from esophageal stricture.

Case XVII. A soldier, aged 30, came to the surgical clinic on Aug. 13, 1906, and stated that he had swallowed an artificial denture on the evening before, which consisted of an incisor tooth with a small rubber plate. Efforts at extraction with forceps were made by the attending physician. The patient had pain on swallowing, slight elevation of temperature. I found definite evidence of emphysema of the skin on the right side of the neck from the face to the third rib, showing that there had been a perforation of the esophagus. For this reason, I refrained from performing esophagoscopy and recommended that esophagotomy be performed in the surgical clinic. As the symptoms indicated it, Kraske opened the neck on the right side and found a perforation of the lateral wall of the hypopharynx. The artificial denture could no longer be found in the esophagus. The bougie entered the stomach without obstruction. Three days later the plate passed off with the

feces. Patient recovered after having a temperature for two days of 38.8 C. and 38.5 C. (101.8 and 101.3 F.).

DISCUSSION.

Dr. Chevalier Jackson, of Pittsburg, Pa., expressed the obligation that all laryngologists are under to Professor Killian for opening a new field, one already fruitful and large with promise. That bronchoscopic work was done at all, and that it was done safely and effectively, was due to him. In his own work Dr. Jackson had had the utmost satisfaction with the tube designed after Dr. Killian's original pattern, which he believed would never be improved in any essential particular. The method of illumination, however, presented some differences. Bronchoscopy with the Kirstein or any other head-light was practical in many but not in all hands. It demanded not only a natural endowment of dexterity, but also such clinical opportunities of practice as come to but few. He had, therefore, added to the Killian bronchoscope a light carrier in a separate tube, and this modification, he believed, rendered the work easier. It could not be hoped to go beyond Professor Killian's own results, but the light carrier at the end of the tube would extend the usefulness of his method by making the instrument effective in the hands of men of lesser skill. Bronchoscopic work for the present concerned itself mostly with foreign bodies, and it was highly desiraable that cases should be dealt with by some one immediately at hand. In every centre of population there should be at least one man competent to explore the bronchi. Every effort should be made, therefore, to devise easier means of work. With the tube which he exhibited any man accustomed to the direct inspection of the larynx might, without hesitation, explore the tracheo-bronchial tree to its second and third subdivisions. In no case of foreign body or exploration for disease was damage done. In no case was shock apparent. The dangers of superior bronchoscopy were those of general anesthesia. The dangers of lower bronchoscopy were even less. His preference was for upper bronchoscopy, as with distally illuminated tubes and the accessory drainage canal in the wall of the tube, the view was as good in a long tube as in a short one. Since the development of Professor Killian's work he could conceive of no foreign body case suitable for expectant treatment. What may be done with the bronchial tube in diseased conditions was a question for the future to determine, but the prospect was bright. He believed that in the near future practically all endo-laryngeal operations will be done by the direct method and that many of the more important examinations will be made in the same way. With the patient in proper position, the larynx thoroughly cocainized, the passage of the tube down to the tracheal bifurcation was so simple a proceeding that it might well be made a matter of routine. A few years ago the diagnosis of simple local lesions below the larynx was very imper-

fect and the treatment was almost impracticable, but Professor Killian's work had shown how to deal with these conditions. Upper bronchoscopy had a considerable field of usefulness as a routine method as an adjunct to the direct examination of the larynx. Where the indication was primarily of tracheal or broncheal trouble, and particularly where operative work was indicated, lower bronchoscopy was a somewhat easier operation. With the tracheotomy done under infiltration anesthesia, he believed it often to be safer. With a separable speculum there was not the slightest difficulty in promptly passing the bronchoscope through the glottis. When he first used Professor Killian's separable spatula he considered it a device second in importance only to the bronchoscope itself. In a modified form he had found it so useful that he was loath to abandon it.

Dr. E. Fletcher Ingalls, of Chicago, wished to add his expression of gratitude to Professor Killian for developing bronchoscopy. There was, however, much to be done to insure the safety of the operation. He had not used Professor Killian's pump for removing mucus from the bronchi, but had adapted Jackson's pump for the ear to this purpose. He emphasized the importance of pulling out the tongue. One must learn from experience the effect of the drugs that may be applied to the mucous membranes for the purpose of preventing bleeding, and how much may be used with safety. There was considerable danger from the use of too much of either cocain or adrenalin. His preference was for upper bronchoscopy, which he would try first, particularly in girls and women, for a reasonable time—not more than fifteen minutes—and if not successful would then do tracheotomy. He had had no success at all with the electric magnet. With a pin-finder something like a corkscrew he had been able to get pins into the middle of the lumen of the bronchus; then to shove the bronchoscope down over them and remove them with forceps. There was danger from the little booklets recommended in Professor Killian's earlier paper, as they might easily become caught in a small bronchus and could not be removed except by tearing out, which would be likely to result in emphysema and death. The swelling of the larynx after bronchoscopy had sometimes required speedy tracheotomy. He cited a case in which he had removed a foreign body by upper bronchoscopy. The following morning the child seemed to be suffocating from swelling of the larynx, he introduced a Dwyer's tube, which was later coughed up, and there was no further trouble. He had seen three cases die after what he would consider easy operations in which he was sure there had been no perceptible injury to the air passages. The chief questions which it seemed important to consider were what causes the danger? How long the operation may be continued? And, what symptoms indicate that the operation is being carried too far?

Dr. A. Coolidge, of Boston, Mass., said that a few years ago if a foreign body were inhaled it was either let alone in the hope that it would be coughed up, or tracheotomy was done and forceps were blindly pushed into the trachea and bronchi, in the hope of seizing it. The mortality of these two methods had not differed enough from each other to establish a principle of action. The first great step in advance was the demonstration that a straight hollow tube could be inserted through a tracheal wound, and the lower trachea and primary bronchi seen by direct inspection. By this simple proceeding, with a comparatively simple armentarium, every surgeon might be ready to successfully reach a large proportion of inhaled objects. Foreign bodies presented a large variety in size and shape, and consequently varied much in the ease with which they might be removed. Every help in the avoidance of injury, time, and the danger of infection might in any case mean the difference between life and death. Consequently a carefully devised series of instruments and a well studied technique were necessary for the greatest success. For this advance in medical efficiency the medical profession and the patient were indebted to Professor Killian. It would be a long time before even a majority of medical men could find within an available distance, when the case arises, either the instruments or the skill to extract a foreign body in the way in which it would be done in Professor Killian's clinic. For several reasons the lower route would be the safer one for a limited equipment, and the question of choice between upper and lower bronchoscopy should properly depend fully as much upon the available equipment both of instruments and skill as upon other conditions. It should always be borne in mind that if a tracheotomy will probably be necessary, prolonged attempts by the upper route might exhaust the patient more than is warranted. The important thing with reference to light is that there should be enough. Until the relative value of different forms of light becomes definitely fixed and the apparatus therefor universally available, there could not be too many resources, if they are efficient. If enough light could be forced into the tube from above without interfering with the work, that was all that was necessary. Practically, however, many in this country had found that the small distal lamp gives so bright a light and is maintained by so simple and portable an electric supply that it was likely to be retained for the present, at least as a substitute. He had not found that secretion shuts off the light any sooner than it would shut off the view of the field if the light came from above. It did add a complication to the tube, and was a source of possible danger by breaking. The light was also more uneven and did not penetrate so far beyond the end of the tube as a powerful light from above. He reiterated what Professor Killian had said, that to do good work, and to avoid injury the instruments must be fitted to the particular case, which meant anticipation beforehand of all possible conditions.

Dr. Frank B. Sprague, of Providence, R. I., called attention to the use of atropine for the purpose of fortifying respiration and drying the secretions. He had employed it successfully in three cases.

Professor Killian, in closing the discussion, said that irritation of a foreign body in a bronchus would very probably cause bronchitis or pneumonia, in which event it was important to understand the general condition of the patient, and whether bronchoscopy should be performed. Some cases were so dangerous that the patient would die unless the foreign body were removed; in such cases the method should be continued and the child be given the benefit of possible relief.

PAPER:

THE QUESTIONABLE INFLUENCE OF SO-CALLED DIATHETIC CONDITIONS IN DISEASES OF THROAT AND NOSE IN CHILDREN.

CHARLES G. KERLEY, M. D.

It will be observed that I have changed the title which was given me by the secretary which reads: "Certain Diathetic Conditions in Their Relation to Diseases of the Throat and Nose." I take it that I was invited to read a paper before this body, in order to bring into discussion the views or observations of one engaged in another line of work than the members of the society and who possibly might see things from a different view point, so that my paper, to be of value, must be essentially a personal one.

In twenty years practice among children of all classes, in hospital, in dispensary, in private among the poor, among the well to do and the wealthy, naturally certain impressions relating to diseased conditions must have been made. The most frequent disorder associated with the nose and throat is enlargement of the tonsils and the presence of adenoids in the naso-pharyngeal vault. So great an etiological influence do these conditions exert, not only over other tissues of the nose and throat but over the entire organism, that I will devote my attention to them alone. This paper is, as I mentioned above, a record of personal observations.

Observation I. The frequency with which these conditions are seen.

Observation II. The distribution of the disorder geographically. The patients are found in large cities, small cities, country villages, in the open country, practically an equal number of children being affected to the proportion of the population regardless of their residence.

Observation III. The distribution of the disorder socially. Hypertrophied tonsils and adenoids are found among all classes, among the very poor and the very rich and those occupying intermediate positions, among those who live well and those who live wretchedly.

Observation IV. The distribution of the conditions among different physical types. The strong and robust, the weakly and those occupying intermediate positions in development and resistance, show the growths with practically equal frequency.

An erroneous impression is to the effect that strong, otherwise well, children do not have adenoids. Some of my patients whom I consider my best specimens of childhood have undergone operations for enlarged tonsils and adenoids. Such a distribution of a disorder as this, it would seem to me, rather negatives the influence of any so-called diathetic state as being a predisposing factor.

Diathesis I understand as meaning "A predisposition toward" a disease. It has not been my observation that any special type of constitutions show any marked tendency toward the development of adenoids or enlarged tonsils. Instead of attributing diseased states of this and other kinds to the vague term of diathesis, it would seem best to try and discover a more rational explanation.

If these diseased conditions occur among all classes of people among the poor and the rich, in the city, in the country, among the well and the delicate, would it not be wise to look for a causative agent in some error in the life of all children, for if it is not based upon a physical defect, it must be due to some error in the life of the individual. What is there then that nearly all infants, regardless of their social position, place of residence or manner of life, do that they should not do? A great majority of the human race during the first years of their lives and many for some time afterward, are habitual suckers, in the use of the thumb, the comfort, the pacifier and other objects less elegant. My attention was

first called to this phase of the matter from noting that the early cases of adenoids, those that were pronounced under the first year, were habitual suckers of the thumb or pacifier. Certain it is that infants and children who indulge in the habit to a considerable extent, show more pharyngeal and faucial congestion than those who are free from the habit. It can be readily appreciated that the vacuum produced through the process of sucking on long objects which extend deeply into the mouth, has a tendency to produce hyperaemia and stases in the delicate blood vessels of the mucous membrane of the parts. Further, the post-pharyngeal space in an infant is a very narrow slit and in many, particularly with high-arched palates, it may be readily understood how the soft palate presses against the posterior wall, and by friction, tends to produce an irritation, congestion and consequent hypertrophy of the lymphoid tissue and an enlargement of the third tonsil. Whether this attempt at an explanation is of value or no, it is certain that habitual suckers developed adenoid more frequently and earlier in life than those that did not have the habit.

What other one condition is there which occurs in all types and classes of children, that may have an influence on the development of the growths? It is the so-called common cold which means a localized congestion or inflammation. All children are subject to frequent colds, those who suffer most are the ones who develop adenoids most frequently. Not only are the colds found in those who have adenoids, but there is a history of colds preceding signs of obstruction. The adenoids return after operations with greater frequency in those who take cold easily, who develop the colds upon the slightest provocation.

What has more to do with the frequent taking of cold in children than any other factor? Years ago I observed and continued observation has proven that the sugar-eating children, those who partake daily of a great deal of sugar, were particularly subjected to colds and that by removing sugar from the diet the tendency would be markedly relieved. If there is an error in the daily life of runabouts that is practiced by all, regardless of social standing, place of residence, etc., it is the excessive sugar eating. This can readily be appreciated when it is realized how readily the appetite for sugar is acquired, how plentiful it is and in what varied ways it can be taken by the child. In a paper read before the American Pediatric Society at Washington, recently, I reported seventy-eight

cases of illnesses in which there was an excess of sugar taking or in whom there was a proven sugar incapacity. In thirty of these there was an involvement of the respiratory tract. It has been found that the sugar susceptibles, those who have but a small sugar capacity, are usually of gouty or rheumatic inheritance and here the proposed so-called diathesis may claim a small part, as it is this type of child who takes cold most readily. By reducing or discarding the use of free sugar, in this type and in others so predisposed, we may after the second year do away with many of the colds and consequently do away with the tendency toward growth of adenoid vegetations.

In regard to the terms scrofulous diathesis and strumous diathesis we hear but little at the present time, the discovery of the tubercle bacillus having relegated them to the position they occupy with thousands of other medical misconceptions. So far as lymphatism or the so-called lymphatic diathesis is concerned it is to be remembered that in all children there is a tendency to hyperplasia of the lymph glands throughout the body both internally and externally. The lymphatic system furnishes in children much less resistance to irritation and infection than is the case with adults, which explains the greater frequency and involvement of lymph structures in children as is expressed in the frequent adenitis (acute, suppurative and tubercular) encountered in the young.

Some children show this lack of resistance greater than others, as is to be expected, so that the condition resolves itself into one of degree. In comparison with adults all children have lymphatism so-called. To say that a child suffers from lymphatism only when the condition is marked might be compared to calling only the severe cases of diphtheria as such, the mild cases being ignored or called by another name. The condition Status Lymphaticus is a distinct pathologic entity and does not concern us in this connection.

To my mind the three factors, the sucking habit, the free use of cane sugar, and the tendency to lymphoid tissue involvement, a condition peculiar to all children, contribute largely to the production of adenoids. The etiological relation of these growths to other disorders of the upper respiratory tract is not in my province to discuss before a body of rhinologists.

DISCUSSION.

Dr. Thomas Hubbard, of Toledo, Ohio, was inclined to believe Dr. Kerley has over-estimated the importance of the thumb-sucking habit, which is not so much a causative factor of adenoids as it is a symptom or an indication that there is present some irritation which established this habit. He agreed with Dr. Kerley's remark concerning the sugar habit as a pre-diposing factor in taking cold. The only satisfactory treatment for children having this habit was to entirely prohibit this article of diet for a few weeks, after which the normal appetite for a variety of foods would displace the pernicious sugar habit.

Dr. J. E. Sheppard, of Brooklyn, was of the opinion that air as dry as that of the average school or home would have more effect in producing colds in the head than would either the thumb-sucking or sugar-eating habit, and that in the line of adenoid prevention it might be the duty of the physician to demand of those who heat houses that they provide apparatus whereby indoor humidity might more nearly approximate the average outdoor humidity than is now the case.

Dr. William L. Ballenger, of Chicago, said the question resolved itself into one of exciting causes and predisposing factors. The exciting causes were the various infections of the upper respiratory tract, particularly in the adenoids and tonsils, and the conditions leading up to the infection were the predisposing factors. There were various predisposing factors other than the ingestion of cane sugar, which was undoubtedly one. While sucking the thumb might have some slight predisposing influence in the production of adenoids, he had never considered it an important factor. In many instances children with no adenoids were thumb-suckers.

Dr. J. A. Stucky, of Lexington, Ky., emphasized the point of the greater toxic effect of cane sugar as compared with any other sugar. He had noticed that among the children of the foundling hospital in the South those who were given no sugar or molasses did not develop adenoids as did those on the regular hospital fare. Sugar undermined the intestinal digestion and predisposed to lymphatic development.

Dr. John F. Barnhill, of Indianapolis, Ind., agreed with the essayist that there is a lymphoid predisposition at this period of life, and with Dr. Ballenger's suggestion that these enlargements are due to infection. Adenoids and tonsils were susceptible to enlargement following the exanthemata, and enlargement of these structures had often been found in association with decayed teeth or with faulty eruption of the teeth. Whatever the predisposing causes might be he believed them to be enhanced by germ producing diseases.

Dr. George B. Wood, of Philadelphia, said that adenoids should not be looked upon as the result of frequent colds, but on the con-

trary they were the most potent cause of rhinitis in children. Further, coryza in children was very hard to diagnose from an inflammation of the pharyngeal tonsil, and the general practitioner often made this mistake. The adenoid tissue of the pharyngeal vault developed before and immediately after birth, and in common with other lymphoid tissues had a distinct function in the manufacture of white blood cells. When the child needed to be protected from infection there was very apt to be hypertrophy of the hemogenic organs, so that it might be that enlargement of the tonsillar tissues was sometimes due to a general infection.

Dr. Perry G. Goldsmith, of Toronto, Canada, called attention to the fact that removal of adenoids did not always relieve the child of colds, and that it was not uncommon to find recurring colds after their removal. He did not agree, therefore, that adenoids were always the cause of colds in children, as one of the speakers had remarked. He thought the operation for adenoids was performed very often simply because some lymphoid tissue was seen with a post-nasal mirror, or when an acute inflammation of this lymphoid tissue was present. Many of these cases did not require operation, and operating in such cases tended to bring discredit upon rhinological practice.

Dr. George L. Richards, of Fall River, Mass., cited two cases which had come under his observation. In one of these the child was not a nose breather, yet it developed a suppurating ear and mastoiditis. Large amounts of adenoid tissue were found in the vault, yet the child had plenty of room for breathing through the nose. In the other case the child was a nose-breather all the time, and when it came to be operated upon because the pediatrist in charge had found that it had repeated colds, a large amount of adenoid tissue was really the cause. In such cases there was infection and hypertrophy of gland tissue which is originally normal and which has a protective office. When adenoid tissue has ceased to be protective it should be removed.

Dr. J. Price-Brown, of Toronto, Canada, thought atmospheric conditions had much to do with diathetic conditions of the nose and throat.

Dr. Sargent F. Snow, of Syracuse, N. Y., had had no experience with children given to the thumb-sucking habit, but he believed the excessive use of cane sugar to be productive of the troubles under consideration. He had given considerable careful study to the matter of taking cold, and the more he looked into it the more he believed that it resolved itself into a question of the quality and quantity of food ingested and of the matter of elimination. As to whether adenoids are produced by infection or by frequently recurring inflammations, he could not say, but he agreed with those who hold that adenoids are usual in children showing a certain predisposition. He was inclined to agree with the view expressed by Dr. Phillips some years ago that hypertrophy of

adenoid tissue follows repeated colds, which seem in some cases to cause a piling up of the lymphatic tissue, in others a purulent discharge.

Dr. Kerley, in closing the discussion, agreed with Dr. Wood's views that the removal of adenoids by no means necessarily prevents the taking of cold, thereby causing no little disappointment to the parents of the patient. He felt that too much was often promised in this respect. As pointed out in his paper, there were other factors in so-called cold than adenoids, one being the very important element of excessive sugar in the child's diet. He cited one of those cases in which the adenoids were said to have been removed three times by different New York laryngologists, and the coryza, more or less persistent, continued. He referred to ten cases that he had reported before the American Pediatric Society at Washington this year, in each of which one or more operations for the removal of adenoids had been performed with but little relief to the nasal symptoms. These children were all sugar eaters in excess and when sugar was removed from the diet, the nasal discharge ceased to a large degree. His personal observation had been that infants who are habitual thumb-suckers develop adenoids more frequently than those who do not have the habit.

PAPER:

HYSTERIA OF THE EAR.

CHRISTIAN R. HOLMES, M.D.

Systematic writers upon hysteria do not lay much emphasis upon the aural manifestation of that disease and yet some of the most important cases, cases in which a mistaken diagnosis may possibly bring about conditions menacing the very life of the patient—are brought to the aural surgeon for diagnosis and treatment. In most of these cases, of course, the hysterical character of the symptoms is obvious upon even a cursory examination but occasionally a symptom complex is encountered which defies a proper analysis except after long and careful observation and there are not a few cases on record in which serious errors in diagnosis subjected the patient to grave surgical operations with all their attendant dangers.

An examination of the numerous clinical histories which can be found in the literature shows that hysteria can simulate every

known organic lesion of the ear with more or less fidelity and has added to the catalogue a long and important series of peculiar aural reflexes in which various parts of the ear play the part of a hysterogenic zone or stigma.

As the pathology of hysteria is unknown it will not profit us to discuss the various theories advanced as they must be left to the neurologist for study and a final determination of their value. What concerns us ultimately, however, is the question of diagnosis. It is on this point that we will be appealed to as consultants and we must be prepared to decide some very difficult problems promptly and with authority. I am not aware that any one has, as yet, attempted a systematic classification of these cases. My study of the subject, however, would indicate that there is a natural classification which would assign to each case its place in one of five catagories. This classification is as follows:

1. Cases in which there is no evidence of any disease of the ear.

2. Cases in which a normal or abnormal ear is the seat of hysterogenic zones—as where certain sounds produce reflex phenomena in distant parts of the body.

3. Cases in which there are abnormal appearances in the ear which can be explained, and which the subsequent history of the case demonstrates as, temporary nervous and vascular phenomena (e. g., angio-neurotic oedematous patches, changes in color, etc.).

4. Cases in which there are slight pathological changes in the ear—real, but insufficient or not of a character to account for the symptoms complained of.

5. Cases in which the hysteric inflicts more or less damage upon the ear for the purpose of exciting sympathy or to induce the performance of an operation by the aural surgeon.

Every physician who has served as an interne in a general hospital or who has had experience with general practice knows how necessary it is to take into consideration all the circumstances —both hereditary and environmental—that surround these hysterical cases, and in the course of our inquiry we will see that in some of the cases submitted to us as specialists in which an hysterical tendency is not suspected by the friends or attending physician it will be the opinion delivered by us that aural disease is certainly not present that will first establish the positive diagnosis of hysteria. It is therefore incumbent upon us to be thoroughly

familiar with all the signs of this neurosis and to bear the possibility of its presence always in mind, especially when we are confronted with cases in which the symptoms bear an obvious disproportion to the physical signs.

Let us now discuss the various phases of aural hysteria in accordance with our classification:

1. Cases in which there are no evidence of any disease of the ear.

Gilles relates a typical case of this kind. A twenty-one year old hysteric for the two years following upon an emotional disturbance was more or less deaf from time to time. The deafness showed itself whenever one spoke to him suddenly. Gilles speaks of it as a paresis of accommodation of the ear. There was no disease of the ear.

Cases of this kind naturally are somewhat rare. In the first place a very large proportion of all the inhabitants of the temperate zone are suffering from chronic catarrhal alterations of the upper air passages and the ears. In the second place practically all hysterics are anaemic and otherwise below normal in resisting power. It follows then that few hysterics will show absolutely normal ears and we can expect to find some slight alterations of a catarrhal character even in cases where we are perfecty satisfied that the aural symptoms are purely hysterical.

2. Cases in which a normal or abnormal ear is the seat of hysterogenetic zones.

These cases are somwhat more frequent than the preceding. They are of several types but probably the most frequent is that connected with sound perception. Steinbrügge has recorded a typical case. An hysterical man of 45 years of age had suffered from peculiar convulsive attacks for 15 years whenever he heard instrumental music. These attacks dated from a severe fright. Other sounds even when they were of great intensity made no particular impression on him. The reflex symptoms included sensations of cold over parts of the body, disturbance of respiration, rotary movements, etc. There was reduced hearing and a chronic discharging otitis media. No hyperaesthesia of the acoustic nerve could be determined.

Voss also records an interesting and instructive case of a character that is much more likely to come under our observation than that of Steinbrügge's. A nineteen year old girl was operated upon for a chronic purulent otitis and mastoiditis. A cutaneous flap was made

from the lower portion of the meatus; the posterior wound sutured and a tampon inserted through the meatus. Change of dressing on the fifth day, removal of sutures, primary healing. There was now a complete hemi-anaesthesia of the body on the operated side, in other words, hysteria. Thereupon the tampon was removed from the ear and not renewed, but merely an outer dressing applied. Prompt recovery.

Voss, in his article on "Diseases of the Ear in Hysterical Subjects," uses this case to illustrate and enforce his contention that packing of the wound cavity after operations for acute or chronic mastoid disease should not be undertaken in nervous or hysterical adults or children. We have all had painful and disagreeable experiences in years gone by with cases of this character and can remember the terrible scenes accompanying the frequent dressings of these wounds. It is indeed fortunate that for some time past we have recognized the positively harmful effects both upon the repair process in the wound and the nervous system of the patient of the old method of tight tamponade, and that we have adopted improved methods of technique that give to the patient less suffering and to the surgeon more satisfactory results more rapidly attained.

3. Cases in which there are abnormal appearances in the ear which can be explained as, and which the subsequent history of the case demonstrates as, temporary nervous and vascular phenomena.

Numerous observers assert that in the course of hysterical manifestations in which the ear is the seat of an alleged disturbance of function that physical changes do occur, passing away without leaving behind them any trace of their existence as soon as treatment of the hysteria results in a disappearance of the aural symptoms. These are important observations and a knowledge of them will help us to avoid what might have been serious errors in diagnosis. Of course no one could fail to recognize the significance of such a phenomenon as a patch of angio-neuritic oedema, an herpetic vesicle or a psoriatic "drop"—but in some cases the changes are limited to a darkening of the color of the drum membrane—it is described as dark gray—or to more or less circummembranous or circum-malleal injection. These cases certainly must be difficult of diagnosis. It is even of record that a perfect picture of myringitis has been presented—without, however, any of the subjective symptoms that go to complete the clinical history

of that disease. Continued observation, conservative and nice discrimination, and a proper appreciation of the nervous phenomena other than those appertaining to the ear, will be necessary to properly guide us in these cases.

It is possible that some cases of vicarious menstruation, where the blood flow is from the ear, should be placed in this class. Gradenigo describes a case occuring in an hysterical girl of 15 years. Blood came from the ear at the menstrual periods whether the regular menstrual flow from the genitalia appeared or not. Removal of small blood clots from the depths of the external auditory canal revealed "six or eight reddish points of the size of the head of a small pin," which Gradenigo concluded were the mouths of ceruminous glands. The drum membrane was absolutely intact. In Shambaugh's case, cauterization of the bleeding point caused the aural periodical hemorrhage to cease, but it is not mentioned in the report of this case whether or not there were nervous or hysterical phenomena connected with the ear symptoms or observed in the intermenstrual period.

4. Cases in which there are slight pathological changes in the ear—real, but insufficient or not of a character to account for the symptoms complained of.

This class includes by far the largest number of cases. As already stated the hysteric is almost invariably below par not only as regards the inhibitory mechanism of her will power, but also as regards the resisting power of her tissues to the ordinary wear and tear of physical existence. Therefore, we find in a very large proportion of all cases certain more or less marked, or more or less serious, lesions of the ear. Diagnosis, of course, is a fairly easy matter in those cases in which the allegations of subjective symptoms by the patient are out of all proportion to the actual findings in the aural system by modern methods of diagnosis—but a certain number of cases present themselves in which the difficulties of diagnosis are very great. I have seen a case, for instance, in which the cerebral symptoms of a fully developed otitic meningitis, in a hysterical girl of 12 years, closely simulated and were regarded as a purely hysterical mental exhibition until within a few hours of death. Even the temperature range is not an absolutely trustworthy measure of diagnosis between the two conditions—especially as the hysteric is so frequently subject to digestive disturbances with intestinal putrefaction and

various signs of an auto-intoxication of which an elevated temperature is not the least frequent. The differential blood count is probably our most certain reliance when the suspected hysterical manifestation simulates an inflammatory otitic condition and it is quite possible that this might have to be repeated a number of times.

We must never forget that many of these patients are quick witted, wonderfully determined to deceive and some of them more than willing to undergo serious operations—if thereby they can secure the attention of doctors and the solicitude of nurses and the anxious sympathy of their friends. From the questions of one physician they learn or infer what symptoms or signs are sought for in the disease that they desire to exploit so that by the time they are consulting their third or fourth medical adviser they are fairly well up in their part and often act it so admirably as to be able to deceive, at least for a time, men of skill and experience.

Voss mentions an hysterical woman who succeeded in having her mastoid opened by feigning a mastoiditis, and then details a case of his own. A woman, 45 years of age, presented herself with the drum bright red and swollen as in acute otitis media just before perforation. There was severe otalgia. He treated her palliatively as a case of acute otitis media for three days and as the condition did not improve performed paracentesis. The tympanic cavity contained nothing but air. In 24 hours the incision closed—no secretion appeared on the occluding gauze but the conditions were the same as before. Voss now made a diagnosis of a neurotic and not an inflammatory condition and prescribed bromides and valerian. In 24 hours the membrane appeared normal and the pain was gone.

It is in this class that we must put most of the cases of hysterical otalgia or hysterical hyperaesthesia of the acoustic nerve. A physical examination of the ear reveals possibly only some slight catarrhal changes—or it will reveal areas of hyperaesthesia or analgesia of the skin in the canal, over the auricle and even down the neck to the clavicular region, the shoulder and the chest wall. These anaesthetic or hyperaesthetic areas should always be diligently sought for as sometimes they are the only hysterical manifestations upon which a diagnosis can be made.

Finally we have 5—Those cases in which the hysteric inflicts more or less damage upon the ear for the purpose of exciting

sympathy or to induce the performance of an operation by the aural surgeon.

It is probably the existence of these anaesthetic areas which permit and induce the hysteric to sometimes inflict upon herself visible injury. Sometimes the injury is practically nil as in those cases where some filthy substance is introduced into the external auditory canal—but, if we were permitted to judge from the standpoint of a normal person, the discomfort would be extreme—even unbearable.

More often, however, there is slight but actual physical damage done. I had a case under my care a few years ago. A young girl about 19 years of age, tall, slender and highly neurotic was brought by her parents on account of neuralgic pains about her eyes and nose. There was a slight posterior hypertrophy of both inferior turbinals and a half dioptric of astigmatism, under homatropin, which was rejected later. When it was evident to her that she was about to be dismissed from further treatment she was brought in great alarm by her parents one morning—her right ear plugged with cotton—with the assertion that the ear had been bleeding copiously during the night as was evidenced by the blood stained condition of her pillow. The cotton taken from her ear was stained with blood, it is true, but an examination of the ear revealed that the latter was normal in all respects. There was not even a slight abrasion. Instituting a search for the source of the blood stains on the pillow and the cotton with which she had deceived her parents, I found several recent abrasions covered with moist blood clot on the right side of the nasal septum. I informed the parents of the conditions and, of course, was met with indigant protests—but the evidence was complete and unimpeachable.

Very recently I have had under my care a somewhat similar case. A woman, aged 28 years, was brought to me by Dr. Duncan, of Burlington, Ky., who was quite convinced that it was a case of hysteria, but wished to have his opinion fortified by an expert examination of the ears, nose and throat. She gave a history of having fallen from a horse seven years before, striking on the right side of the head. She was unconscious for twenty-four hours. Two years ago she had headaches and a pain in the right ear followed by a discharge of pus. With the pus were also discharged at various times pieces of bone which aggregated in all

about a teaspoonful. Other pieces of bone were discharged from the ear into the pharynx and were coughed out and a couple of pieces were also passed out through the right nostril. This astonishing statement was corroborated by her husband who stated, as conclusive evidence, that *he had seen the bones.* Since then the ear had not given her any trouble until two weeks ago when there was sudden and severe earache followed by a displacement of the lower jaw to the left and tenderness and retraction of the neck muscles on the right side. All movements of the head were painful, and on account of the displacement of the jaw, the mouth could not be closed and saliva was constantly drooling. The patient entered my consulting room the very picture of abject misery—pale, haggard, stooped—the head retracted to the right, barely able to walk across the floor and almost unable to talk. I was almost willing to believe her statement that she had tasted neither food nor drink for two weeks. The physical examination revealed an exquisite tenderness over the right mastoid but otherwise a perfectly normal ear. I agreed perfectly with Dr. Duncan in the diagnosis of hysteria and determined to try the power of suggestion. I assured her positively that she would speedily recover from her distressing symptoms, admitted her to a room in my hospital and sent her husband home. After admission to the hospital she refused to take her internal medicine (Elix. Sod. Brom.), alleging her inability to swallow—although later she was found to have drunk water out of a glass accessible to her. After she had fallen asleep, quite firm pressure made over the sensitive mastoid elicited no response. On the second day the nurse reported that the right ear was discharging freely—an examination, however, revealed another trick. She had endeavored to fill the external auditory canal with a thick muco-purulent secretion which she had probably coughed up and introduced with her fingers,—the inner half of the canal and membrana tympani were found dry and normal. Up to this time there was total deafness of the right ear both for voice and tuning work. The treatment consisted of a mild current of electricity to the ear, leeches over the mastoid and suggestion. At the end of five days all of her severe and disagreeable symptoms had disappeared, her hearing was completely restored and she returned home cured and wonderfully improved in appearance.

I have been surprised in my study of the literature to find so few cases of this character. Cases of simulated or of functional deafness, of reflex neurosis and curious aberrations of hearing, are abundant but cases in which a deliberate attempt was made by mechanical means to induce the patient's family and friends to believe that a serious disturbance of the ear was present are very few.

One of these cases from the literature, I will recite to you, in the first place because it is really a typical case of this character and in the second place on account of its quaint phraseology and its shrewd analysis of the phenomena presented. It is found in a curious old volume published in Breslau, in 1726, under the title of "Sammlung von Natur und Medicin wie auch hierzu Gehörigen Kunst und Literatur Geschichten. Frühlings Quartel." This old book is written in a curious mixture of German and Latin and printed in a still more curious mixture of black letter and Roman type. The following is a translation of the author's account of the case: "Another case is reported by Dr. Joh. Pet. Wahrendorff of Liebnitz; a girl of 20 of phlegmatic temperament who passed pebbles through the ears, nose, mouth and uterus; and finally, sand through the bladder" (he describes the sand as like that used by the German servants for scouring floors). "The pebbles resembled so closely those found in the creeks and fields and were so unlike anything found in the human body that he thought it must be a case of fraud. On visiting said patient a small pebble protruded from the left ear and the patient was sneezing violently. The parents, who were honest and pious people, assured him that these paroxysms of sneezing continued until the offending pebbles were forced either from the nose or ears. If the pebbles occurred in the mouth there was violent coughing and finally coughing with ejection of the pebble. If the pebble was to be passed per uterus or vaginam the patient complained of pain and pressure in the abdomen. Menstruation regular every fourth week. The color and size of the pebbles differ, some are black and others white, large and small, depending on the part of the body from which they come. The parents had collected quite a large number of these pebbles, but they did not collect or save the sand. The suggestion of witchery had been made but as the parents, who are very pious people, had assured the doctor that there was no deception and no witchcraft and as the pebbles so closely resembled those found in the fields and

creeks, the doctor was very much puzzled. In addition to the sand the patient's urine had at various times assumed different colors, red and bloody, then black as ink and finally green. Two of the pebbles from the ear weighed five grams each, one was white, the other black. One from the mouth which was black weighed nineteen grams. One from the anus, white in color, weighed one drachm two scruples; another, one drachm; this one was also white. One from the vagina, ten grams. After a careful examination of the pebbles the following conclusions were reached:

1. That they were ordinary and real pebbles.

2. That they could not have been generated in the human body but certainly must have gained entrance from without.

3. That the same must necessarily have entered the body in their present shape and size, through the act of deglutition just as they leave the body through the excretions; and this being so the patient must know for a certainty when and how these pebbles reached her abdomen.

4. * * * *

5. Had they been carried from the stomach through the blood into the emunctories, this could not have occurred in the ears, nose, vulva and urethra without laceration of the meatus and without hemorrhage.

6. If they had been placed into each "emuctorium externe" this is not so strange, because it would be easier to do by the human hand, than for a supernatural power to do so.

7. It is to be remarked that the power (which did it) acted in a sensible human way for those in the nose and ears were small; those in the anus and vulva large; and none at all in the eyes; and if the devil had done this and had acted in a supernatural way, he would not have followed this order and would certainly have lacerated the external meatuses. The author finally concludes that the girl did this herself—despite the parents' statement to the contrary. Later he concludes that if the girl had been bad enough to do this she undoubtedly colored her urine by drinking various colored inks of her father's, he being a teacher and requiring inks of different colors."

The diagnosis of these cases often presents unusual difficulties and I have been taught that it is unwise to make a hasty diagnosis of hysteria in those cases, occurring as they so frequently do in neurotic subjects, where the initial examination reveals no ade-

quate cause for, say, a persistent and earnest allegation of pain in and about the ear. We are arriving now at a wider and more accurate knowledge of the pathology of the nose and its accessory cavities and we know that it is quite possible for a diseased ethmoid cell, or sphenoid cell, or for an hypertrophied middle turbinate, to produce reflex disturbances in the ear so out of all proportion to the apparent local disturbance that the real cause of the ear trouble can be overlooked even by careful observers, for a long time. I recall one case occurring in a young girl who alleged pain and paraesthesia in the ear and who was examined by me repeatedly with a negative result. She was of a neurotic temperament and passed through the hands of one neurologist after another. She had an ocular muscular disturbance which was corrected by tenotomies and glasses. Hereditary syphilis was suspected in her case and she was energetically treated. Finally it became slowly and increasingly evident that she was suffering from an ethmoiditis and the inflammation of the accessory cavity with its extension to neighboring structures gradually unfolded itself as the cause of the aural disturbance which was advancing into a confirmed chronic catarrhal deafness. And yet the original diagnosis of hysteria seemed to be justified up to the time of the discovery of the infected ethmoid cell. In another case, that of a middle aged lawyer, persistent pain in the mastoid without any visible lesion was diagnosed originally as hysterical mainly on account of the neurotic temperament of the overworked and worried patient. Long continued observation of the case finally convinced me that there must be a real underlying pathological cause. I opened the mastoid and found an extensive osteo-sclerosis. The otalgia was banished by the operation and has not returned since.

We see, therefore, that in some cases, indeed in many cases where the diagnosis of hysteria would seem to be natural and inevitable almost at once, it is incumbent upon us to search diligently, conscientiously and repeatedly for any condition either of the ear itself, the nose, the throat or the accessory cavities, that might be able to account for the symptoms complained of, not only for the sake of properly understanding the patient's condition and benefitting the patient, but for the sake of our own reputation.

The management of these cases, whether they be cases of pure hysteria or real aural disease with an element of hysteria is always a delicate matter. Should we make a diagnosis of hysteria in a

patient with a normal ear or an ear with but very slight pathological changes I hold that it is our duty to turn the patient over at once to a neurologist, acquainting him with all the facts in our possession—the case is one for a nerve specialist for the patient has no aural disease. In those cases in which there is more or less disease of the ear it becomes a matter of nice discrimination as to how it should be dealt with. Where the hysteria is severe and the aural disease slight I would prefer again to turn the case over to a neurologist and undertake only such treatment of the ear at such times and under such circumstances as in his judgment was best, for we must never forget that continued treatment of the ear will tend to confirm such illusions as the patient may have concerning its condition.

Where, however, the condition of the ear is one calling for immediate attention we must give it that attention—probably in such a case operative, or at least mechanical—bearing in mind the hysterical state of the patient, always speaking cheerfully and, possibly, stretching the facts a little to furnish a favorable prognosis for the disturbed mind. In all events it is imperatively necessary for us where extensive operative wounds have been inflicted as in an operation for mastoiditis to reduce the discomforts of the period of repair to the minimum and to hasten the process of repair to the utmost. Fortunately modern methods of dressing and handling these cases permit us to do this. All painful packing can be omitted and by proper grafting the area of raw surface is very materially reduced. But throughout all of these procedures we should always bear in mind that in reality we are but doing emergency work on some one else's patient—and this hysterical patient, who has illusions concerning the ear which are beyond the realities which we have recognized, operated upon and healed, should be put in the care of the neurologist at the earliest moment at which we can conscientiously discharge her.

It is certainly unnecessary before a body like this to argue against the propriety of undertaking or pretending to undertake an operation on the ears of these patients for the purpose of attempting to "satisfy their minds." One might characterize this as trivial folly were it not possibly fraught with grave and even serious consequences. I would not mention it were it not that one occasionally sees in the surgical press reference to cases of such unjustifiable childishness. Such a procedure even if alleged

to be successful, does not assist in restoring the will power, the mental resistance of the unfortunate patient—on the contrary, it but confirms her in her belief in the reality of the hysterical symptoms under consideration and paves the way for fresh illusions as to the same or other parts of her body for the relief of which she will demand other similarly successful operations. I only mention the matter, gentlemen, for the purpose of expressing an unqualified condemnation.

In every volume of the special journals devoted to the diseases of the ear one can find records of cases of aural hysteria. I have therefore confined my bibliography to a few special cases selected from these journals and to the important and interesting cases that I have found in the leading general journals or in monographs.

BIBLIOGRAPHY.

GRADENIO. Klin. Vortr. aus d. Geb. d. Otol. u Pharyng. Rhin. Laryng. von Haug. 1896, I, 13. Sulle manifectazioni auricalari del l'isterismo.

ANDRÉ BOUYER; FILS. Contribution a l'étude de l'hysterie troubles cérébraux d'apparence otogène chez les malades parteurs d'algies mastoidiennes. Paris, 1903, 31 p.

A. BOUYER. Contribution à l'étude de l'hysterie troubles cérébraux d'apparence otogène chez les malades parteurs d'algies mastoidiennes. Paris, 1903, 1, 577; 609.

BARTH. *Deutsch Med. Wchnschr.*, 1900, XXVI, 22. Zur Symptomatologie der hysterischen Taubheit.

E. BARTH. Manifestations of traumatic hysteria in the organ of hearing. *Arch. Otol.* N. Y., 1903, XXXII, 443-447.

S. SZENES. Hysterie im Anschluss an Otitis media acuta. *Ungar. med. Presse,* Budapest, 1902, VII, 273-274.

F. SCHULTZE. Ein Fall von hysterischer Taubheit. *Deutsche Aerzte-Ztg. Berl.*, 1901, 73-75.

VOSS. Ueber Ohrenleiden bei Hysterischen. *Ztschr. f. Ohrenh.*, Wiesb., 1901, XI, 1-44.

G. KING. A case of hysterical deaf-mutism. *The Laryngoscope,* 1900, IX, 341-348.

J. THANISCH. Ein Fall von hysterischer Taubheit. *Arch f. Ohren. Leipzig.*,

RIBALKIN. Vertigo auralis hysterica. *Bolnitsch Gaz. Botkina,* St. Petersb., 1899, X, 1591-1601.
1905-6, IXVI, 116-119.

LANOSIS & MARCHADOUR. *Ann. d. mal. de l'orcille du larynx, etc.,* 1889, XXV, 10. De la Surdite hysterique vraie.

A. WESTPHAL. Ueber einen Fall von hysterischer Taubstummheit, *Charite-Ann.* Berlin, 1899, XXIV, 602-607.

J. VEIS. Die hysterische Taubstummheit. *Muenchen. Med. Wchnschr.* 1899, XIVI, 415.

D. GRANT. A case of hysterical nerve deafness with spontaneous recovery. *Journ. Laryngol.* London, 1898, XIII, 281-283.

F. P. HOOVER. Hysterical deafness and report of cases. Manhattan Eye and Ear Hosp. Rep. N. Y., 1897, 61-63. Also *Med. Herald,* St. Joseph, 1897, XXVI, 205.

J. F. Shepherd. Three cases of hysterical mastoiditis. Tr. Am. Laryngol. Rhinol. & Otol. Soc. 1896, N. Y., 1897, 11, 49-53.
Kretschmann. Hysterie unter dem Bilde von Taubheit. *Arch. f. Ohrenh.* Leipzig, 1897, XLII, 288-.90.
Hartmann. Dysbasia hysteria cum otalgia hysteria. *Verhandl. d. deutsche Gesellsch.,* Jena, 1896, V, 110-112.
W. T. Van Dyck. Hysterical (or functional) deafness. *Brit. Med. Journ.* London, 1895, i, 973.
E. Lamphear. Hysterical deafness and aphasia in the male. *Alienist and Neurol.* St. Louis, 1895, XVI, 460-463.
E. F. Ingals. Hysterical aphonia and deafness. Tr. Pan-Am Cong. 1893, Wash., 1895, Pt. II, 1570-73.
T. Kako. Taubheit der Hysterischen. *Ztschr. f. d. Med. Gesellsch. zu Tokyo,* 1894, VIII, 13 H. 6.
J. P. Wahrendorff. Von einem Magdlein in Schlesien so aus Mund Nasen, Ohren und Vulva ordentliche Kiesselsteine soll von sich gegeben Neben. *Samml. v. nat. u. med. Gesch.* Breslau, Frühlings Quartal, 1726, 487-492.
Habermann. Auftreten von Hysterie und hysterischer Schwerhörigkeit im Auschluss an eine Otitis media catarrhalis acuta. *Prag. Med. Wchnschr.,* 1880, 233.
M. V. Uspenski. Hysterical deafness . *Laitop Khirurg Obsh. v. Mosk.,* 1882, V. 3-5.
G. L. Walton. Deafness in hysterical nomianaesthesia. *Brain.* London, 1883, V. 458-472.
A. H. Williams. Hysterical Deafness. *Guy's Hosp. Gaz.,* 1889, ns. III, 163.
Steinbrugge. Die otiatrische Untersuchung eines an mannlicher Hysterie leidenden Kranken. *Ber. d. oberhess. Gessellsch. f. ..Nat. u Heilk.* Giessen, 1890, XXVII, 164.
H. V. Wurdemann. A case of hysterical deafness. *Med. News,* Phila., 1891, LVIII-186.
Allbut Syst. Med. Vol. VIII-1899. Hysterical Deafness.

DISCUSSION.

Dr. S. MacCuen Smith, of Philadelphia, said it was not uncommon for these patients to go the rounds of the gynecologist, the genito-urinary man,the ophthalmologist, and finally the otologist and neurologist. Two or three visits were generally necessary before such patients could be convinced that there was nothing the matter with them. He cited the case of a patient who complained of severe pain over the mastoid and of tinnitus. The tinnitus was that of the ticking of a watch held about two inches from the ear, and was heard distinctly in both ears. Upon examination nothing was found in the nasopharynx or throat, and yet she complained so of mastoid pain that she was admitted to the hospital and kept under observation for about a month. She was about to be dismissed as a purely neurotic case, when she developed a temperature, and complained of pain more intense over the right side. The right mastoid was opened and extensive necrosis found. She made a prompt recovery, but a week later it was necessary to open the left ear, when the same condition was found. The tinnitus could not be accounted for. It seemed to be synchronous with the heart beat, and when the temperature rose the ticking went up to 130 per minute.

Dr. Wells P. Eagleton, of Newark, N. J., said Dr. Holmes did not emphasize the point that there is a great deal of difference between malingering and suffering from hysteria. It was his belief that hysterics actually suffer. The man or woman who put pus in the ear, for example, was a simulator, and very different from one who has hysteria. It was a dangerous thing to make a diagnosis of hysteria. Within the last year he had had three cases come to him with a diagnosis of hysteria. In one such instance the patient complained of vomiting and noise in the ear. He had been treated in one of the hospitals for hysteria, but when he examined the man he found beginning optic neuritis which went on to total blindness.

Dr. J. E. Sheppard, of Brooklyn, recalled three cases of hysterical mastoiditis which he had reported to this society some years ago, the discussion of which brought out the fact that a number of the speakers had seen similar cases, some of which were operated upon and some not. Since that time he had seen a number of cases. As a rule they did not develop real tenderness over the mastoid, and all had failed to show evidence of active inflammatory trouble in the ear. The most striking case of the kind that he had seen was that of a young girl in her early twenties who had had one ear operated upon three times and the other twice for mastoiditis. She complained of tenderness, and was anxious to have the other ear operated upon the third time. She was a hospital case and he had been sent for to do an immediate operation. Finding that she had no temperature, and that the skin over the mastoid region was macerated, he sent her back to the ward, where she was kept for three weeks without developing any symptoms. Sometime after she was dismissed from the hospital she returned, complaining of pain so intense that she could not sleep. She was then sent to a neurologist, who could do nothing for her with bromides, etc. Such cases were always more prevalent after newspaper reports of mastoid operations, and were also seen where others of the family or friends had been operated upon.

Dr. Wendell C. Phillips, of New York City, would divide all so-called hysterical cases into two classes: First, cases of true hysteria, and second, those that have gone beyond the stage of hysteria and are really insane. One class of patients with imaginary conditions could be convinced, at least temporarily, that such conditions do not exist; others could not be convinced no matter what arguments might be employed. The latter class were insane. He had seen a number of such patients, and believed that this broad classification should always be borne in mind. He had seen particularly within the past year, a number of cases of hysterical mastoiditis. In one such case he had yielded to the temptation of employing the "blood-clot" method, with perfect success. He cited the case of a woman who came to his clinic with the statement that she had a bedbug in her ear. He found nothing upon examination,

but could not convince the patient of this fact. Deciding that the only way to dispossess her of the idea was to put in a bug and take it out again, he succeeded in finding a bug, which he inserted cautiously against the drum. When he drew it out and showed it to her she was greatly relieved, and he had not seen her since.

Dr. Max A. Goldstein, of St. Louis, cited a case, previously reported, of a neurotic young woman of twenty, who had a peculiar form of hemorrhage from the ear, which extended over a year. Every possible feature of the case which might have had a bearing on the hysterical phase was eliminated—vicarious menstruation, malingering, etc.—and, as a last resort, the patient was sent to the hospital and watched closely for three days and nights. Finally her head was placed in a plaster cast for two days, the patient having been told that an operation would be performed. The external meatus was packed with gauze and the plaster of paris bandage applied. After removal of the case there was no further bleeding, and the hearing which had been *nil*, was normal.

Dr. Walter B. Johnson, of Paterson, N. J., referring to the question of temperature, which some of the speakers seemed to consider indicative of the presence of diseases, said he had seen an hysterical case run a temperature of 109° with no evidence of any kind of disease. The patient finally fell into the hands of a gynecologist who made an exploratory incision, sewed it up, and the patient was absolutely relieved thereafter. He cited several cases which he had treated successfully by means of suggestive therapeutics. It was a question, however, how far this matter of suggestion should be carried. He believed that these hysterical patients do really experience suffering.

Dr. Holmes, in closing the discussion, emphasized again the fact that the diagnosis in these cases presents unusual difficulties, and should never be made hastily. It was probable that the subject would be cleared up by a better understanding of the various reflex disturbances and by further study of the pathology of the accessory sinuses.

PAPER:

A STUDY OF EAR SYMPTOMS IN ARTERIO-SCLEROSIS, WITH SPECIAL REFERENCE TO THE LABYRINTH.

JOHN J. KYLE, M.D.

For a number of years, we have firmly believed that the early and late ear symptoms, as observed in general arteriosclerosis, are worthy of greater consideration, not alone as disagreeable complications of this growing disorder, but as early and important symptoms in the diagnosis of the disease. My conclusions have been verified upon those suffering from arteriosclerosis in both middle life and old age. Since having our attention directed in this channel by experience and also by the contributions on the subject by Alexander,[1] in 1902, an instructive monograph, entitled "Arteriosclerosis of the Labyrinth and Nerve Centers," by E. Escat,[2] of Toulon, has appeared.

Otological literature is singularly quite barren in regard to the ear symptoms in arteriosclerosis. I can accentuate an opinion quite freely expressed and believed, that many ear symptoms, too often considered as purely local, are the local expression of a general disease. Therefore, a tinnitus and slight deafness, with slight loss of bone conduction, may be of far greater significance than simply diseases of the perceiving apparatus, and for this reason, we should go deeply into the etiology of slight deafness, possibly thereby blocking the march of a general disease, which may ultimately harass, if not annihilate the individual.

Progressive loss of bone conduction in individuals of advanced years, has heretofore usually been considered as being due to structural change in the bone, that is, a sclerosis, the result of age. I cannot but believe that this is somewhat fallacious and that the loss of bone conduction is primarily due to change in the blood vessel walls, and secondarily, to a nutritive change in the temporal bone from faulty blood supply.

We frequently find bone conduction normal in people of advanced age who are free from arteriosclerosis. However, in forty

consecutive cases with typical pipe-stem arteries, varying in age from sixty-two to one hundred years, inmates of the National Home for Disabled Volunteer Soldiers, Marion, Indiana, loss of bone conduction was present in every case. The general average bone conduction, T. C. 512, normally heard for 20″, was 7″ in the right ear, and 8″ in the left.

To make myself perfectly clear in the beginning, we do not propose to consider arteriosclerosis as an accompanying condition of old age, as in the class mentioned, but confine our remarks to the disease as observed in the productive stage of life, that is, in individuals ranging in age from thirty to fifty years.

The youngest case of arteriosclerosis examined showing loss of bone conduction, was aged twenty years. The diagnosis of general arteriosclerosis was made by Dr. Wynn, of Indianapolis.

The theory of sound conduction is quite thoroughly discussed by Boenninghaus,[2] Archives of Otology, October, 1906. The transmission of sound waves through the conducting apparatus, especially by mass or molecular movements, is interfered with if there is a sclerosis of the arteries of the temporal bone. The hardened arteries cannot take on a similar and simultaneous movement with the surrounding bony structures and in consequence, the movement is retarded and bone conduction is thus correspondingly diminished. There is also a corresponding faulty metabolism of all the bony structures, corresponding to the amount of disturbance in the arteries of the temporal bone and middle ear. The perceiving apparatus may undergo a similar and probably earlier degeneration contemporaneous with the arterial change in the basilar artery, which will be explained in a consideration of the pathology of the subject. The intensity of air and bone conduction depends primarily upon the density of the bony structures, which is controlled by the blood supply, and secondarily upon the perceiving apparatus.

For a better understanding of the relationship between the ramifications of the arterial blood supply and the auditory nerve in its peripheral and central endings, it is necessary first to refer briefly to the anatomy of the nerve.

The auditory nerve takes its origin in the bipolar ganglionic cells within the internal ear, joins the brain at the lower border of the pons varolii, and subdivides into two parts, the vestibular and the cochlear nerves, which envelope the restiform body. The

cochlear nerve, which is the true nerve of hearing, sends its fibers to the lateral cochlear nucleus and the ventral cochlear nucleus, a ganglion in close relationship to the restiform body. Fibers from the ventral cochlear nucleus pass to the periphery and terminate in the gray cortex of the superior convolution of the temporal lobe, supposedly in the middle of the first temporo-sphenoidal convolution. In this region, posterior to the perceptive center of hearing, is also situated the center for auditory images. The anterior part of this center, according to Alt, quoted by Escat, responds to high pitched tones, and the posterior, to low pitched tones. Fibers from the lateral cochlear nucleus pass parallel to the fibers of the ventral cochlear nucleus and end in the corpora quadrigemina. According to Gowers,[4] certain fibers decussate about the nuclei and pass from each ear to the opposite hemisphere, thus a disease of these centers may produce a transient deafness in the opposite ear.

The vestibular branch, the nerve of equilibration, passes into the superior vermis of the cerebellum, ending in the nuclei of Deiters, and, according to Cunningham, also in certain sensory nuclei of other cranial nerves, as the lingual, vagus and glosso-pharyngeal.

The artery supply of the labyrinth has been very thoroughly described and illustrated by Siebenmann, Politzer and Shambaugh.[9] The labyrinthine artery, the single artery of the labyrinth, is a branch of the basilar artery, and is a part of the central brain system, susceptible to the same pathological changes as observed, for illustration, in the artery centralis retinæ. Branches are given off to the cochlea, vestibule and semicircular canals. Delicate convolutions of the arteries supply the scala tympani, the scala vestibuli and the cochlear spiral, as well as the macula acustica utriculi and sacculi and the semicircular canals, and are thus in minute relation to the auditory nerve and its entire subdivision, the convolutions of the veins and their convergence into one large vein, the vena canaliculi cochlear, resembling thus, the artery.

According to the investigations of Shambaugh, no blood vessels were found by him in the organ of Corti. Blood vessels appear, however, in the connective tissue supporting the membrana basilaris. The basilar membrane is supposed by Boenninghaus, to vibrate by molecular movements communicated to the labyrinth waters by the stapes, and its function is probably controlled by the

increased or diminished blood supply to the supporting connective tissue.

Escat has given us a careful description of the cerebral blood supply and its intimate relation and influence upon the aural centers

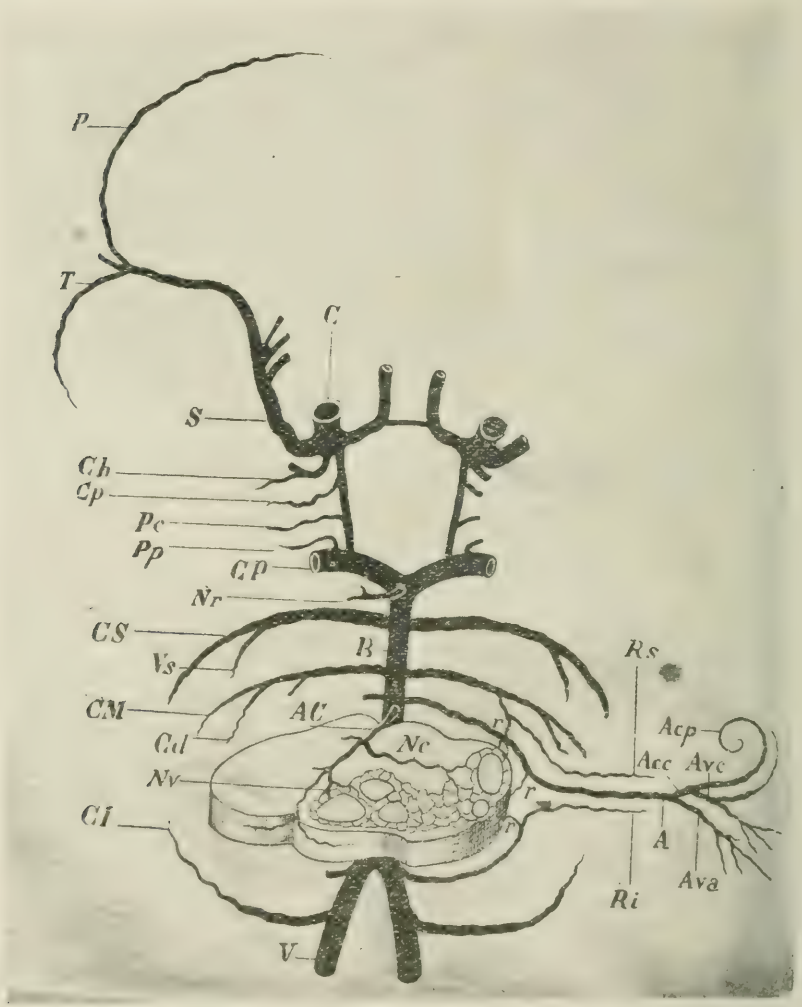


Fig. 1. Scheme of circulation of arteries of labyrinth and brain. (After Escat.)

C. Left internal carotid artery. B. Basilar artery. V. Left vertebral artery. CP. Left posterior cerebral artery. S. Sylvian artery. P. Branch of the Sylvian artery supplying the ascending parietal convolution, in which is the center of conscious equilibration. T. Temporal branch of the Sylvian artery, which is the nutritive artery of the temporo-sphenoidal convolution, the center of auditory perception and auditory images. Ch. Branch of the communicating posterior; Cp. Anterior choroid—both of which traverse the posterior arm of the internal capsule through which pass the acoustic tracts. Pc. Branch of the posterior communicating; Pp. Branch of the posterior cere-

and the labyrinthine terminal nerve fibers. A close study of Figure 1, will show the possibility of functional and organic change in the different centers from circulatory disturbances.

The classical causes of arteriosclerosis are syphilis, laborious occupation, alcoholism, lead poisoning, infectious fevers, auto-intoxication, vaso-motor disease from central disurbances, heredity, lack of balance of ductless gland secretion.

It is unnecessary for the elucidation of the subject, for me to do other than refer to the subject of interstitial myocarditis and valvular insufficiency as sequential or secondary to arteriosclerosis, other than to express the theory accepted by most writers, that fibrous myocarditis is but one of the expressions of a general or local arteriosclerosis.

Heredity is recognized as an important factor in the etiology of arteriosclerosis. The same rule may be applied to arterial change as to hereditary influence in catarrhal deafness as referred to first by Von Troeltsch, and to otosclerosis by Siebenmann, Dencker, Bezold and Koerner.

Arteriosclerosis may be local or general in character and thus a sclerotic change in the arteries may be limited to the ear alone, and be unilateral or bilateral in character.

In the writer's experience, syphilis is a very frequent cause of labyrinthine deafness. We are sometimes prone to believe that sudden loss of hearing, especially with partial loss of bone conduction, is due to some condition resembling Ménière's disease, rather than to disease of the labyrinthine artery. Syphilis more often brings about a primary change in the vessel walls, and the change may be endarteritis obliterans, arteritis, and fibrous degeneration of the wall. The fibrous deposits in the walls of the arteries were regarded by Thoma as nature's method of repair following degeneration of the muscular tissue of the artery. In deafness from

bral—both of which supply the cerebral pedunele, by which pass the bulbo-cerebellar cerebral labyrinthine tracts. Nr. The central artery, of which a collateral branch furnishes a terminal artery to the red nucleus, which is a relay of the cerebello-cortical paths. CS. The superior cerebellar artery. CM. The middle cerebellar artery. CI. The inferior cerebellar artery. Vs. Branch of the superior cerebellar artery, which is the principal supply to the superior vermis, in which is the center of reflex equilibration. Cd. Branch of the middle cerebellar artery, which supplies the dentated body, in which is the center of reflex equilibration. AC. Central artery of the medullar group, supplying the principal medullar centers of hearing. Nc. Branch to the cochlear nuclei. Nv. Branch to the vestibular nuclei. Rs. The superior radical artery of the auditory and facial nerves (the nutritive artery of the auditory trunk). Ri. Radical auditory artery (nutritive of the auditory trunk). rrr. Bulbar branches of the radical artery and the internal auditory. A. The internal auditory artery (nutritive artery of the labyrinth). Acc. The common cochlear artery (of Siebenmann). Avs. Interior vestibular artery (nutritive artery of the vestibule). Avc. Vestibulo-cochlear artery (nutritive artery of the inferior spiral of the cochlea and of the vestibule). Acp. The proper cochlear artery (nutritive of the two superior spirals of the cochlea).

syphilis, we also frequently find present a beginning general arteriosclerosis and mild interstitial myocarditis. The deafness may be entirely confined to one side. In general arteriosclerosis from age, deafness is usually bilateral. Typhoid fever and many infectious diseases are followed by circulatory disturbances, sometimes for many months, and if the patient is not carefully guarded against these conditions which might retard repair, local or general change in the arteries and veins may occur, very obscure at first, and very insidious.

Pearce and Stanton,[6] *Journal of Experimental Medicine,* 1906, demonstrated on rabbits that a sclerotic area in the aorta and aneurismal bulgings, due to degenerative changes and calcareous deposits in the muscular fibers of the media, resulted from the injection of 1 to 1-1000 solution of adrenalin every other day. If these results hold good in man, it stands to reason that the promiscuous and continued use of adrenalin in nasal irritations, and the continued use of the drug hypodermatically for the relief of asthmatic conditions, should be discouraged somewhat until our knowledge of the subject has been more thoroughly defined.

The pathology of general arteriosclerosis is well understood and a brief reference to the changes observed may be all that is necessary. The affection probably begins as a structural change in the vaso vasorum, and is fibrous in character. In consequence of this, the nourishment of the artery wall is disturbed and connective tissue degeneration takes place in the media. Fatty degeneration soon follows in the intima with the formation of deposits of calcereous salts. The vessel wall is narrowed and sometimes obliterated.

The pathological change noted in the labyrinth is of more special interest. The same general rule of change takes place in the labyrinthine artery and its branches as in the larger arteries. The terminal arteries in the labyrinth are so small that any structural change is quickly manifest in the nerve structures. As soon as the nutrition of the basilar membrane and organ of Corti is partially or completely cut off, there is an atrophy of the sensory auditory cells and connective tissue proliferation of all the structures. The same change may be observed in the nerve endings of the vestibulæ and semicircular canals.

A study of the schematic drawings in Figure II., taken from Alexander and Escat, will give the reader a clear conception of

the change in the cochlea. Figure A represents the normal organ of Corti. In Fig. B, there is shown a complete atrophy of the cells of Claudius and Deiter, and the epithelial cells of Reissner's membrane.

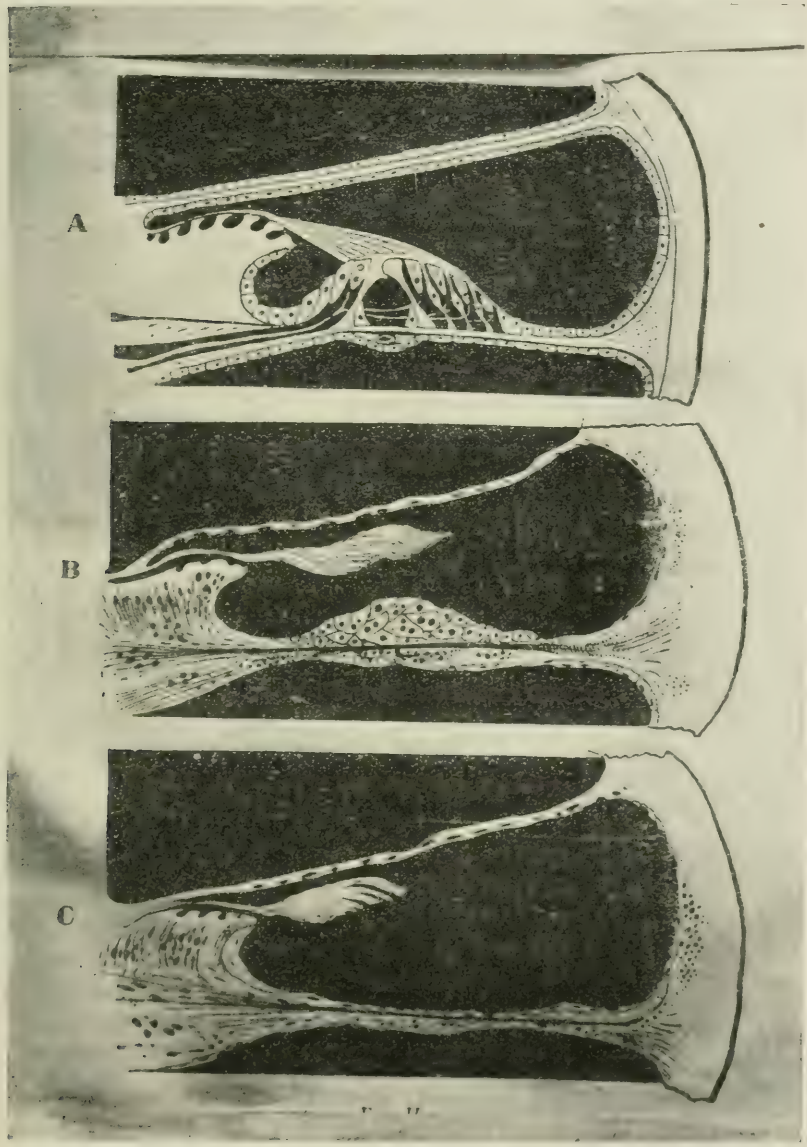


Fig. 2. Schematic Drawing showing the Normal Organ of Corti in comparison with the Organ of Corti in Arteriosclerosis. (After Alexander and Eseat.)

The cells of the organ of Corti have undergone connective tissue proliferation, with closure of the tunnel of Corti and disappearance of the vas spirale. In Fig. C, there is represented the last stage of the destructive process as described by Aleaxnder, that is, absorption of the nerve elements and connective tissue deposits.

The change in the brain structures from arteriosclerosis may vary according to the amount of nutrition carried to the parts. In endarteritis obliterans in the vessels to the center of hearing and equilibration, there is, on account of the slow change in the artery wall, degeneration and atrophy of the brain cells. The same morphological change takes place when the blood supply is partially destroyed. In hemorrhagic extravasation from rupture of the atheromatous blood vessels, there is death of the cells. Thus alteration in the center for audition and conscious equilibration, or equilibration, must vary according to the blood supply and inherent vitality of the cells.

The general symptoms of arteriosclerosis are increased arterial tension, increased tortuosity and prominence of the arteries of the temple, hypertrophy of the heart, and if the last is present, there is generally a lowered vitality of the individual, a sensation of age, and tiring, as from overwork, followed by an appearance of premature aging. In interstitial myocarditis, associated with sclerosis of the coronaries, the pulse may be extremely weak and irregular, and the apex beat of the heart very strong. In other cases, the pulse may be full and the heart beat loud and displaced to the left. According to Osler, in addition, "The combination of heightened blood pressure, a palpable thickening of the arteries, hypertrophy of the left ventricle, and accentuation of the aortic heart sound, are signs pathognomonic of arteriosclerosis."

Analysis of the urine usually shows increase of the urates and long, thin, hyalin casts, undergoing granular degeneration. Other kidney changes will become manifest in arteriosclerosis of the kidney, as albumen, uric acid and calcium oxalate crystals. Many other symptoms could be enumerated. For further information, the reader is referred to the contributions by Stengel[4] on the subject of myocardial disease.

The ear symptoms which should direct our attention to a local or general circulatory disturbance, are unilateral or bilateral tinnitus, slight and progressive deafness, loss of air and bone conduc-

tion, dizziness sometimes early in the disease and in the later stages of the disease, sometimes hallucinations of hearing. The ear symptoms necessarily vary according to the extent of the sclerosis. It is well known that in severe hemorrhage or progressive anemia, deafness and tinnitus may occur. If the hemorrhage and anemia are severe and prolonged, deafness may be permanent, being due to an obliteration of some of the blood vessels to the cochlea or the special cochlear center in the brain. A passive tinnitus or a passive dizziness is probably due to a circulatory disturbance in the arteries of the semicircular canal or special centers of equilibration. These conditions are frequently attributed to liver disorder, rather than a valuable symptom of a possibly beginning general arteriosclerosis. A deafness without dizziness, tinnitus, or Ménière's syndromes, with some general symptoms of arteriosclerosis, is indicative of thickening in vessels to the nucleus, and if associated with peripheral paralysis, may be indicative of disease of the internal capsule. Loss of hearing with dizziness may be due to a vascular disorder from tumors in the cerebrum, cerebellum or pons varolii, as well as to a sclerosis of the labyrinthine artery. Loss of hearing and dizziness continuing for a long time, are valuable signs of vascular disturbance in the entire encephalon. If the eye symptoms are also present, the evidence is complete.

A beginning sclerosis of the vertebral artery may press upon and irritate the cervical sympathetic fibers which send fibers to the labyrinth, supposedly through the non-medulated cells of Remak, found by Erlitzki, in the auditory nerve (Bonnier), probably producing a tinnitus or dizziness long before the sclerosis has extended to the arteries of the labyrinth.

Escat calls attention to the sympathetic system and its influence, especially in the production of the serous effusion in Ménière's disease, which may take place in the labyrinth, from a sudden irritation in the vaso-dilator fibers or paralysis of the vaso-constrictors. It must be conceded that any molecular change in the blood vessel wall, as described by Cohnheim, from intrinsic or extrinsic causes, predisposes to Ménière's disease.

The diagnosis of local or general arteriosclerosis depends upon the symptoms enumerated above. The essential point to be accentuated in this paper, is the early symptoms of arteriosclerosis as manifested in the ear, that is, tinnitus, vertigo, nutritive change in the membrana tympani, and the necessity for a careful general

examination of such cases. The diagnosis of faulty nerve perception, with the modern tuning fork, is easy. The location of the lesion in the nuclear or peripheral endings of the nerve, is somewhat difficult, however, Gradenigo[5] has quite well established the theory that there is diminution in the bone conduction for highest tones in peripheral nerve deafness, whereas in central deafness, there is not as pronounced loss of perception for high tones as for low ones. In both central and peripheral deafness of slight or pronounced degree, it is reasonable to presume a corresponding loss of bone conduction for all tones. Before making the above differential test, it is necessary to exclude any disease of the conducting apparatus. In disease of the conducting apparatus and nerve deafness, the test for central deafness is difficult.

In a late case of aphasia under observation, there was a diminution in low tones on the left side, as well as high tones, a further proof of the theory of Gradenigo.

The disease should not be confounded with Ménière's disease, which is a condition sudden in its onset, though probably often due to primary arteriosclerosis, hyperæmia of the auditory nerve from ingestion of certain drugs, hysterical deafness, or nerve deafness from toxic absorption, as is sometimes observed in parotiditis, or hemorrhagic extravasation into the labyrinth, from a fall or blow upon the head.

For want of time and space, the middle ear symptoms will not be considered, other than to call attention to the diagnostic value of the presence of the arcus senilis of the tympanic membrane, present in so many cases of general arteriosclerosis.

In a general sense, the prognosis depends upon the extent of the sclerosis, that is, whether or not it is general or local in character, and the degree of arterial change.

If habit, occupation or disease affecting the trophic and vasomotor systems can be controlled etiologically, before the disease is far advanced, the prognosis may be favorable. Those cases due to syphilis which are discovered early before degeneration has taken place in the blood vessels and nerve of audition, in which the disease is purely functional rather than organic, the prognosis under treatment, is favorable.

When the cells of the basilar membrane or of the organ of Corti are once destroyed, they cannot be restored. It is unfortunately impossible to prognosticate this condition for the reason that here-

tofore too little attention has been given to the affection by the otologist. These cases have drifted to the general medicine man, who has more often ignored the ear symptoms and given them no recognition in the diagnosis of general disorders.

I believe it is just as essential for the neurologist to study aural symptoms as eye symptoms in brain disease. The ear is as much an off-shoot of the encephalon as the eye.

There are also certain aural symptoms when taken separately or collectively, that may enable us to tell satisfactorily something of the cure or relief of the deafness, dizziness or tinnitus. As to the value of many of the symptoms in prognosis, I am indebted to the illuminating remarks of Escat.

If tests of hearing show slight nerve or central deafness rather than labyrinthine deafness, the prognosis is more favorable. Involvement of the centers of equilibration may respond to treatment. Prognosis in Ménière's syndromes, due to arterial change, is dependent upon the amount of destruction in the artery to the semicircular canal, and if slight, prognosis is favorable, whereas if severe, the prognosis is unfavorable.

Persistent dizziness with slow pulse, respiratory disturbances and paralysis, is a grave symptom. The prognosis is unfavorable in continued deafness with Ménière's syndromes. When vertigo and deafness are transient and variable in character, the lesion is probably in the center of equilibration, and the prognosis is favorable.

With the establishment of complete labyrinthine deafness, accompanying tinnitus may suddenly disappear. A lesion in the center of equilibration, due to arterial change or arteritis obliterans, may possibly be relieved in time by compensatory circulation.

That the syndrome is intimately associated with change in the walls of the central blood vessels is emphasized by the frequency with which these symptoms precede hemorrhage into the internal capsule and their frequency in the chronic form of meningitis, so that when associated with increase of blood pressure, tinnitus, vertigo and impairment of hearing have become recognized as of extreme importance in the fore-shadowing of the graver intra-cranial disaster.

The treatment of arteriosclerosis of the ear is both general and local, depending somewhat upon the exciting cause. Those cases with hereditary influences as predisposing factors, do not respond

to treatment as do those with syphilis or acquired causes. However, in both conditions, the iodide of potassium, in from two to five grains, four or five times daily for long periods of time, is indicated.

H. Richardson[7] recommends Glycolate of Sodium, in five grain doses, in arteriosclerosis from any cause.

Where the heart is able to withstand the shock, hot baths are indicated. Too much exercise is contraindicated in any stage of the disease, and rest is more essential than exercise.

The diet should be carefully regulated. The patient should only consume a moderate amount of meat, no alcoholic liquors, and very little tea, coffee or tobacco. A moderate amount of water should be taken, the excessive use of water being discouraged.

The question of the effort to control the high blood pressure observed in some cases, due to the presence of toxines, must be governed by the etiology in each separate case. In the nephritic cases, high pressure is supposed to be beneficial, whereas in cases of angina pectoris, apoplectic or Ménière's syndromes, those drugs considered as vaso-dilators, may be beneficial.

The local treatment consists in inflation of the middle ear twice or three times weekly, through the Eustachian catheter with superheated air, for the stimulating effect of the air, and external massage. If there is a suspected sclerosis of the blood vessels of the Eustachian tube and middle ears, the treatment differs but little from that for dry catarrh.

BIBLIOGRAPHY.

1. Alexander. *Archiv. f. Ohrenh., Leipzig,* Aug. 1902.
2. Boenninghaus. *Arch. Otol.* Oct. 1906.
3. Escat. Arterio-sclerose en Labyrinthe et des Centres Acustiques.
4. Gowers. Diseases of the Nervous System.
5. Gradengio. Handbuch der Ohrenheilkunde, Vol. II.
6. Pearce & Stanton. Experimental Medicine, 1906.
7. Richardson. *New York Med. Journ.,* Sept. 30, 1906.
8. Stengel. Arteriosclerosis as a General Disease, Medicine, June, 1906.
9. Shambaugh. Blood Vessels in the Labyrinth of the Ear.

PAPER:

VISUAL DISTURBANCES SHOWING A CASUAL RELATION TO DISEASE IN THE SPHENOIDAL SINUSES, WITH THE REPORT OF A CASE, TOGETHER WITH CORONAL AND SAGITAL SECTIONS DEMONSTRATING THE RELATIONS EXISTING BETWEEN THESE CAVITIES.

JOHN W. MURPHY, A. M., M. D.

It is not my intention, in this paper, to go into the history of suppuration in the posterior ethmoid and sphenoid cells, as since the first report of suppuration in these sinuses in 1872,[1] to the present time the literature is prolific on this subject. This is especially the case since 1885, when Schaeffer began to call our attention to the symptoms and treatment of the disease in the living. Nor do I wish to go into an analysis of all of the symptoms which may manifest themselves in disease of the sphenoid and ethmoid cells. I simply wish to confine myself to the report of a case which I have met with recently, in which the eye symptoms were pronounced, and the causal connection seemed traceable to disease in the spenoidal cells. This causal connection was also confirmed by necropsy. I also wish to exhibit some specimens which were made during my investigation into this subject, and which will illustrate the close relation existing between the contents of the orbital cavity and the ethmoid and sphenoid cells.

The intimate relation existing between these two cavities, and what effect disease in the one can have upon the other, must always be a question of much importance to the rhinologist and ophthalmologist.

That these cavities are more often the seat of disease than we suspect is constantly being forced upon us by the revelations of the post-mortem room. The suppuration is usually caused by the severer forms of acute rhinitis, while certain forms of influenza seem especially prone to attack the sphenoid cells. There seems

to be no question but what sphenoidal and ethmoidal suppurations are more frequently met with now than formerly. In the European clinics of Chiari and Lichtwitz, only about 2 per cent of the cases show sphenoidal suppuration.

In the examination of 600 cases in the Massachusett's General Hospital, Rogers,[3] found these cells diseased in 5 per cent of the cases. My experience leads me to believe this higher per cent is more nearly correct for our country.

That the sphenoid is rarely diseased alone but nearly always in conjunction with one or more of the other accessory cavities of the nose seems to be the experience of all observers.

The difficulty of inspecting or even probing the normal sphenoidal opening, which is situated just above the posterior end of the middle turbinate, no doubt accounts for the low percentage of recorded cases. In a number of cut sections in my collection the anatomical relation of the posterior end of the middle turbinate, or the superior turbinate and ethmoid bulla, render the opening exceedingly difficult, if not impossible, to enter with the probe during life. The average depth of the opening is about 7.5 cm. from the anterior nasal opening, and the probe passes in at an angle of 35 degrees with a line perpendicular to the nasal opening. As you can see upon the cut section, the anterior wall of the sphenoid forms the upper edge of the choanae and by keeping this point in mind, as suggested by Gruenwald,[4] the normal opening may more readily be located by the probe.

From the close connection existing between the optic nerves and the cavernous sinuses, with the sphenoidal sinuses, separated as they are by such thin bony walls, or even by a membrane only, it seems strange that disease in these cells is not more often manifested by pronounced eye symptoms. I think we are beginning to recognize more and more that many cases of retro-bulbar neuritis of obscure cause may have their origin in a suppurating sphenoidal cell. This is especially true if the optic neuritis is unilateral in character. Since I have been investigating the subject more closely, I feel quite certain that a causal relation did exist between several obscure cases of optic neuritis, seen in the past, and a sphenoidal suppuration, but I did not connect them at the time.

Dr. C. R. Holmes,[5] has recorded a number of such observations, in which there could be no question of the causal connection of the eye symptoms and a suppurating sphenoidal cell.

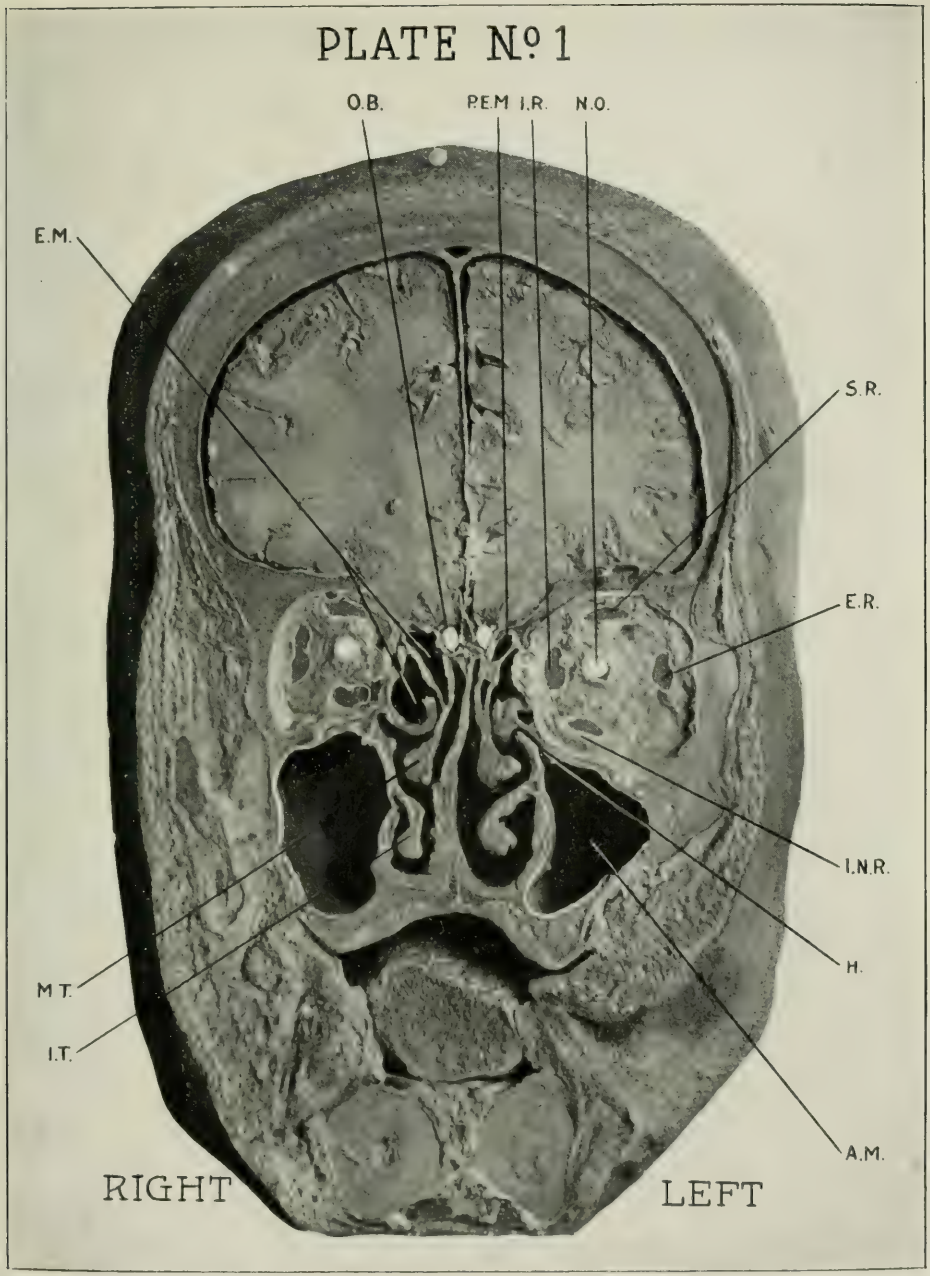


PLATE NO. 1.

Frozen Injected Coronal Section, showing the relation of the posterior ethmoid cells to the orbital and cranial cavities. I. T. Inferior Turbinate. M. T. Middle Turbinate. E. M. Ethmoid Cells. O. B. Olfactory Bulb. P. E. M. Posterior Ethmoid Cell extending over the sphenoid sinus. I. R. Internal Rectus Muscle. N. O. Optic Nerve. S. R. Superior Rectus Muscle. E. R. External Rectus Muscle. H. Hiatus Semilunaris. A. M. Antrum Maxillaris. I. N. R. Inferior Rectus Muscle.

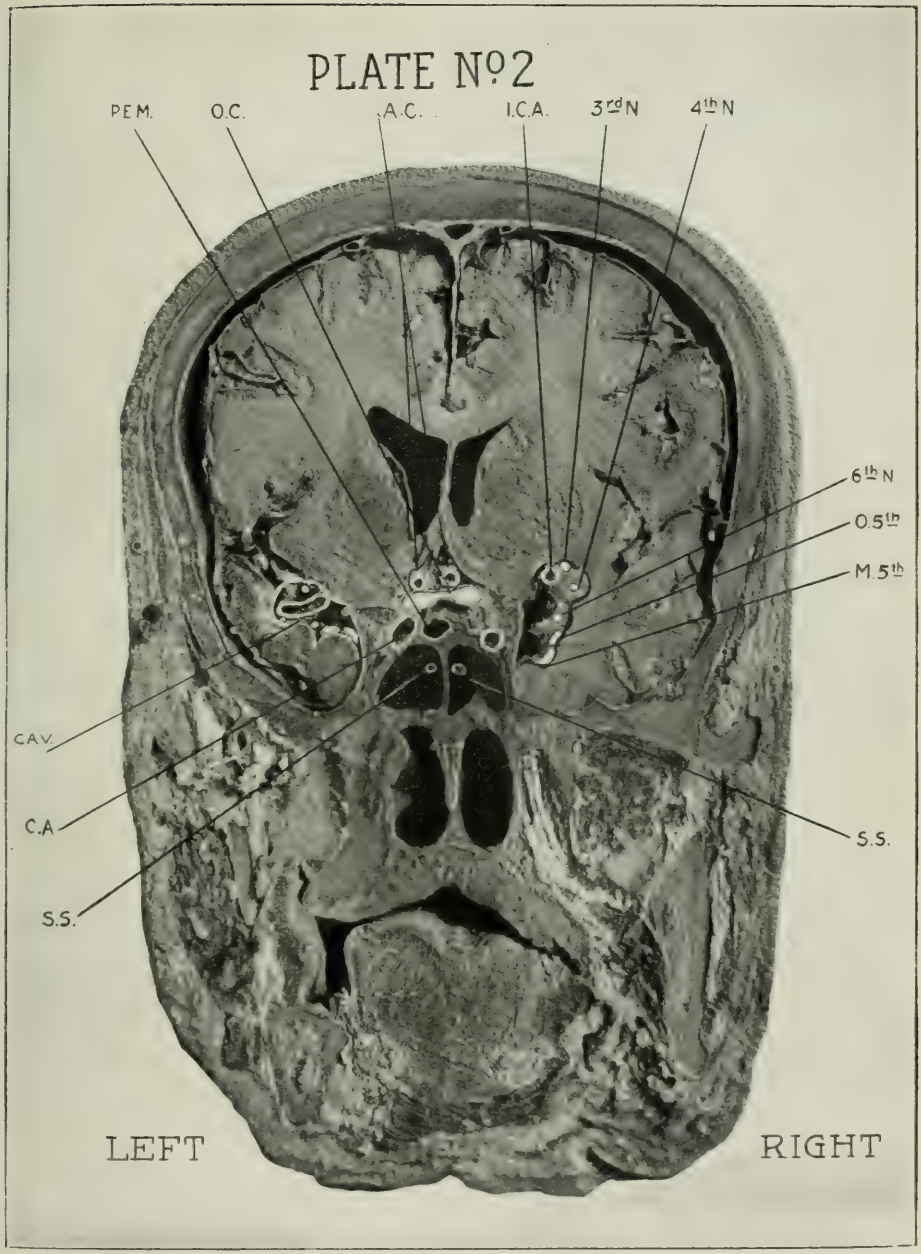


PLATE NO. 2.

Injected Frozen Coronal Section through the optic chiasm and the cavernous sinus, showing the relation of its contained vessels and nerves. S. S. Sinus Sphenoidalis, right and left, showing ostium Sphenoidalis. P. E. M. Posterior Ethmoid Cell extending back over the sinus. O. C. Optic Chiasm. C. A. Carotid Artery in Bony Canal. I. C. A. Internal Carotid Artery in Cavernous Sinus. 3rd N. Third Nerve. 4th N. Fourth Nerve. 6th N. Sixth Nerve. 5th O. Opthalmic Division of Fifth Nerve. 5th M. Sup. Maxillary Division of the Fifth Nerve.

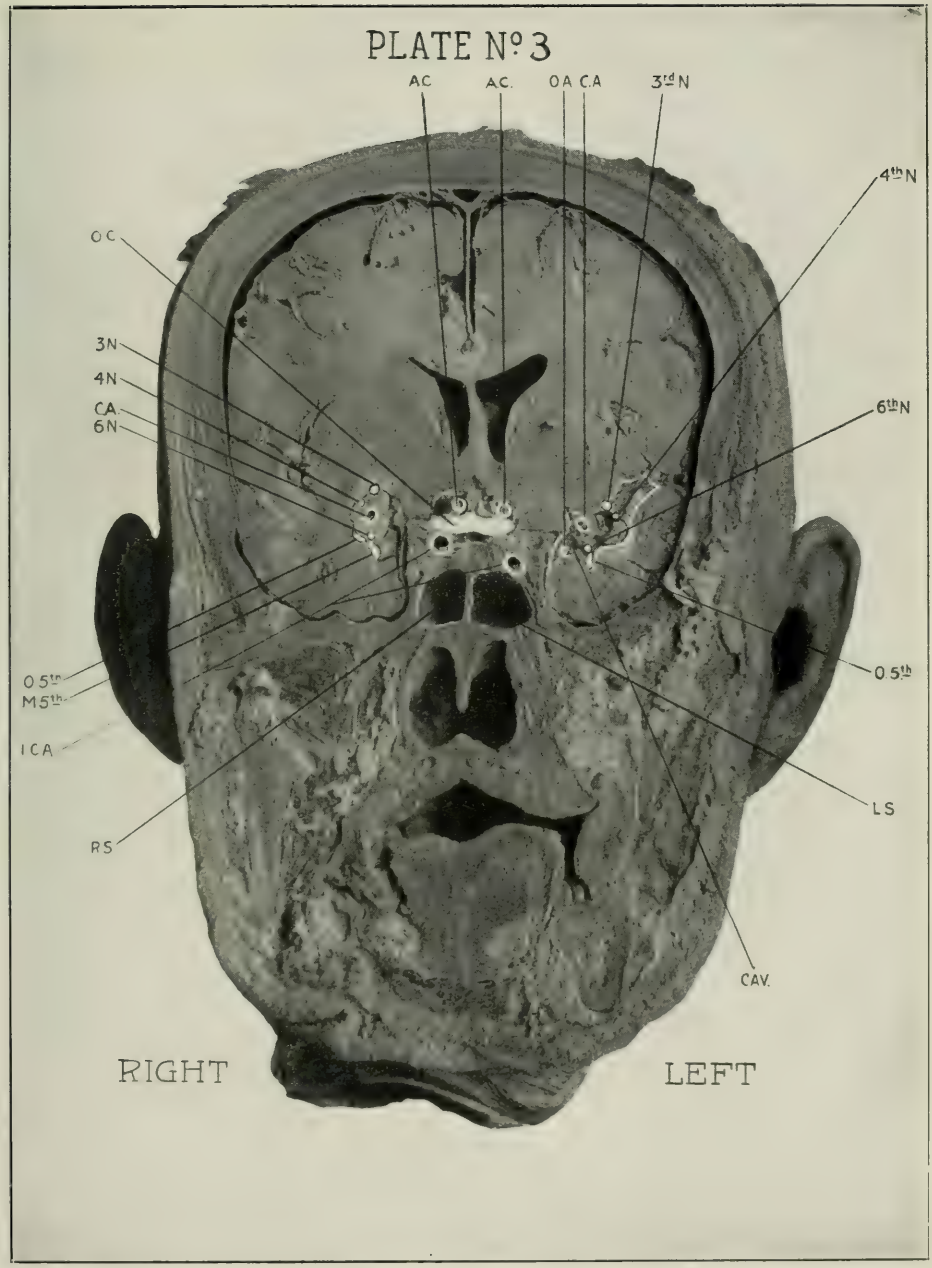


PLATE NO. 3.

Injected Frozen Coronal Section through the Cavernous sinus just back of the optic chiasm, showing the relation of the sinus to the sphenoidal cells and the position of the arteries and nerves in the cavernous sinus. C. A. V. Cavernous Sinus. C. A. Carotid Artery-Internal. 3rd N. Third Nerve. 4th N. Fourth Nerve. 6th N. Sixth Nerve. O. 5th N. Ophthalmic Division of Fifth Nerve. L. S. Left Sphenoid. R. S. Right Sphenoid. I. C. A. Internal Carotid Artery in Bony Canal of Sphenoid. M. 5th. Sup. Maxillary Division of Fifth Nerve. O. C. Optic Chiasm. A. C. Anterior Cerebral Artery, right and left. O. A. Ophthalmic Artery.

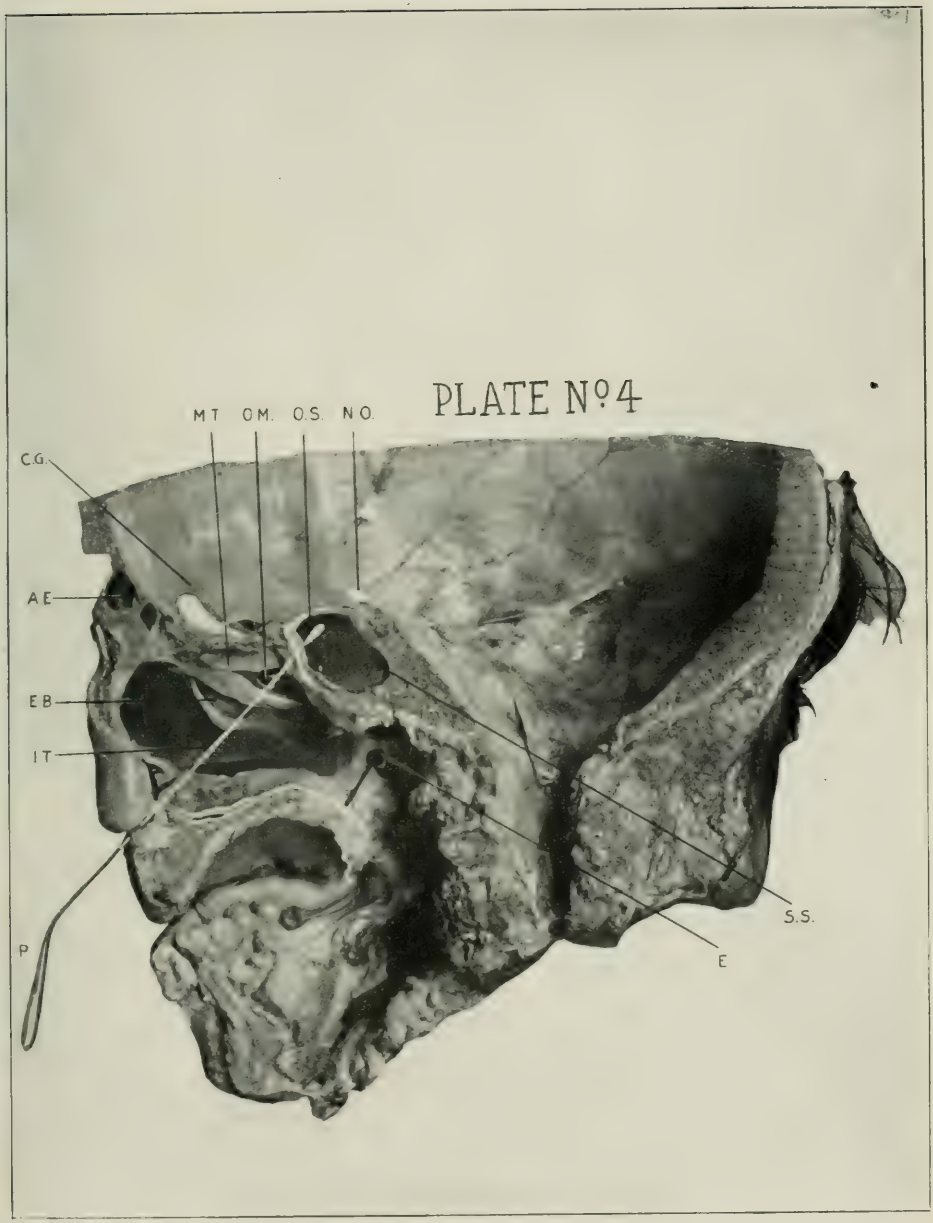


PLATE NO. 4.

Sagittal Section, showing the relation of the sphenoid with the optic nerve. Turbinated bodies atrophied. S. S. Sinus Sphenoidalis 30 m.m. long; 25 m.m. broad; 20 m. m. deep. E. Bougie in Eustachian Tube. N. O. Nervus Opticus. O. S. Ostium Sphenoidalis. O. M. Ostium Maxillaris. M. T. Middle Turbinate atrophied. A. E. Anterior Ethmoid Cell. E. B. Ethmoid Bulla. I. T. Atrophied Inferior Turbinate. P. Probe passing through Ostium Sphenoidalis.

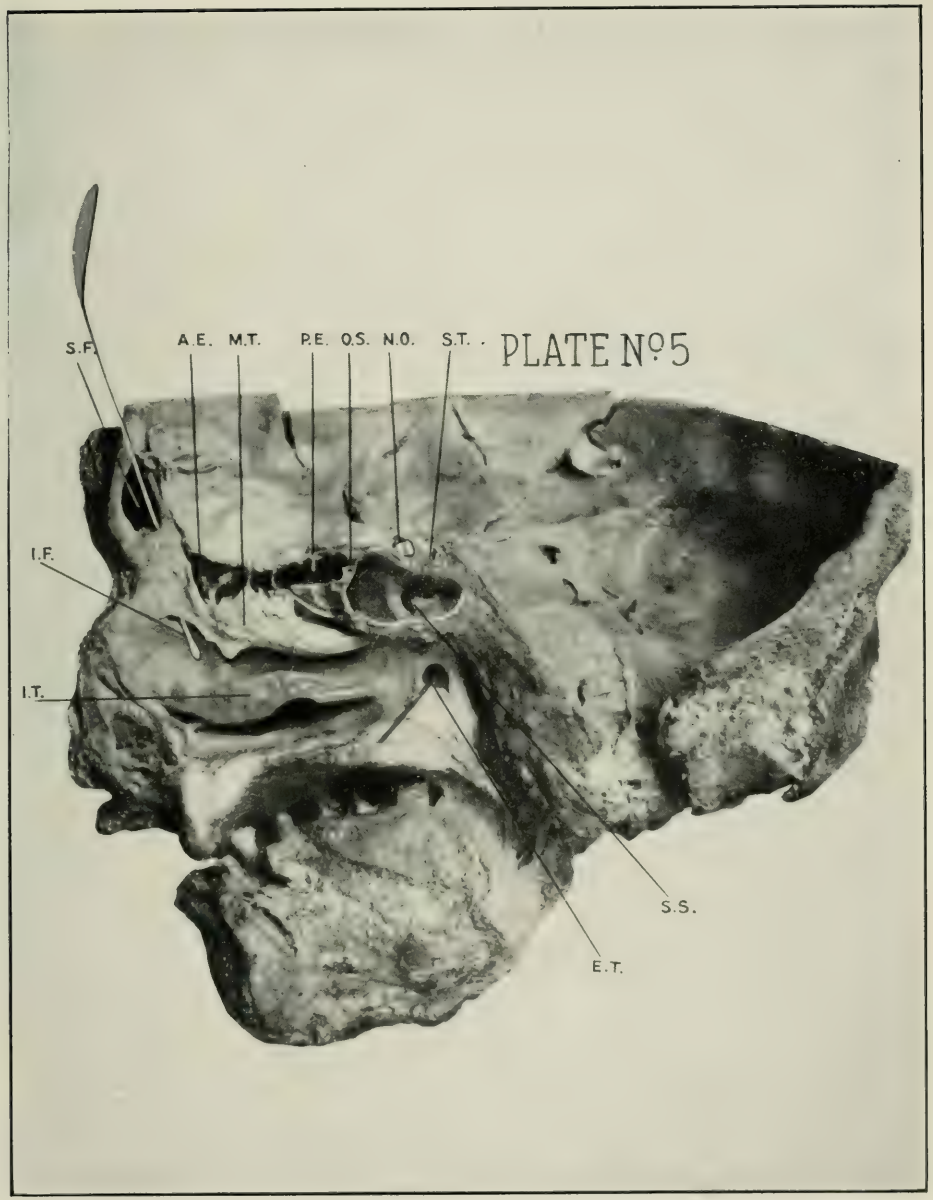


PLATE NO. 5.

Sagittal Section, showing the relation of the sphenoid with the optic nerve and the ethmoids with the frontal sinus. S. S. Sinus Sphenoidalis. E. T. Bougie in Eustachian Canal. S. T. Sella Turcica. N. O. Nervus Opticus. O. S. Ostium Sphenoidalis. P. E. Posterior Ethmoid Cells. M. T. Middle Turbinate. A. E. Anterior Ethmoid Cell. S. F. Sinus Frontalis. I. F. Infundibulum with probe passing from the Frontal Sinus. I. T. Inferior Turbinate.

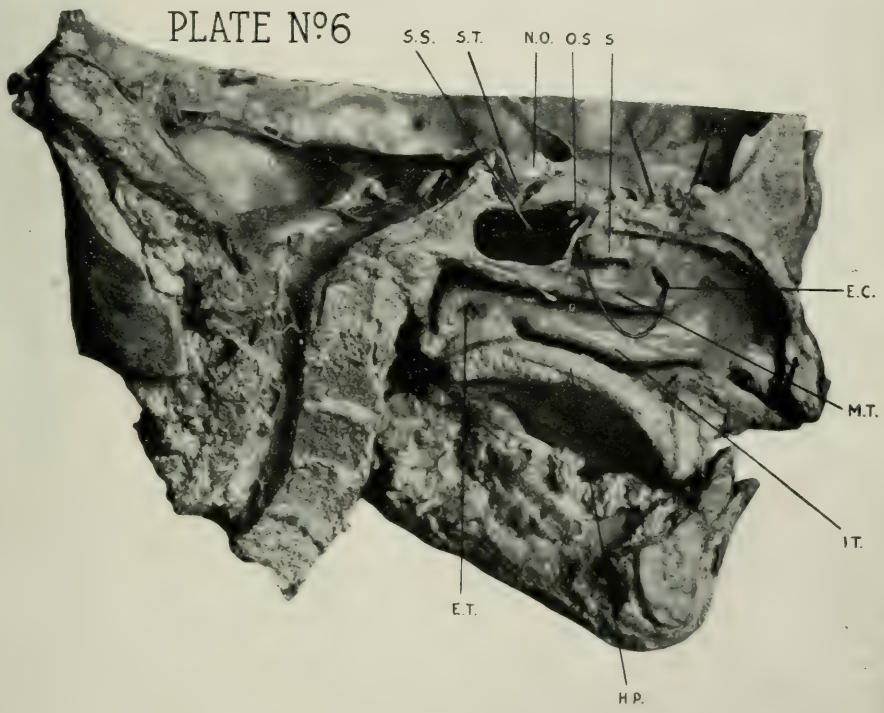


PLATE NO. 6.

Sagittal Section of the left half of the head, showing sphenoid and ethmoid cells. E. T. Eustachian Tube. H. P. Hard Palate. I. T. Inferior Turbinate. M. T. Middle Turbinate. E. C. Ethmoid Cell in Middle Turbinate, with probe passing back into sphenoidal opening. S. Superior Turbinate. O. S. Ostium Sphenoidalis. N. O. Nervus Opticus. S. T. Sella Turcica. S. S. Sphenoidal Sinus.

The fact that the veins of the orbit discharge the greater part of their blood through the ophthalmic veins into the cavernous sinuses, which is only separated from the sphenoidal cells by the thinnest of bony walls, or even by a membrane only explains why an inflammation in the one can easily extend to the other and produce all of the symptoms of a retro-bulbar neuritis.

I feel that our knowledge with regard to the network of veins and lymph passages of these cavities is far from perfect, and I feel certain that the more we investigate the relation, the more often we will be able to find a causal connection between certain eye symptoms and diseased sphenoidal or ethmoidal cells.

The number of such cases reported each year is far in excess of those of the past, and I hardly think it can be due to an increased zeal in diagnosis, but rather to a failure on our part to make a diagnosis. As Gruenwald[6], says, "Ophthalmic troubles, such as asthenopia, scotoma, painful irritation of the ophthalmic nerves, and deterioration of writing power as noted by Schaeffer, are explicable by the neighborhood of the orbital and the basal nerves. Pain is usually more violent in acute inflammation, but then it is, on account of radiation to more distant regions, still more uncertain as a diagnostic sign. The same holds good with disease of the ethmoid bone, only where the troubles are usually more severe than in any other cavity, and here, more especially, that disagreeable complication of constant pain, mental depression and intolerance to nervous excitement may be found. Asthenopia is also frequent, and the pain will be referred to the region of the eyes."

In order to get the experiences and opinions of various ophthalmologists as to the causal relation between loss of sight, or optic neuritis, due to disease in the sphenoid and ethmoidal sinuses, Prof. Onodi[7], submitted to a number of them a series of questions on this subject. From their answers you will see that even in the large eye clinics of Europe, a causal relation is not commonly recognized.

[1]Prof. Leber, says: "Although I have turned my attention for many years to the connection between the diseases of the posterior sinuses of the nose, and those of the organs of sight, I have been able to note very little worthy of record with regard to the sphenoidal sinuses and the posterior ethmoidal cells."

Schmidt-Rimpler: "My experiences afford me no proof that empyema of the sphenoid cavities can of itself cause an affection of the nerves."

Prof. H. Sattler: "Unilateral optic neuritis and optic atrophy are in no way characteristic of disease of the sphenoid and ethmoid cavities."

Prof. Axenfeld: "It is my opinion that diseases of the optic nerves, (neuritis, pressure atrophy) are, if we except actual tumors and perforating cases, much rarer than we might expect in diseases of the sphenoid."

From these reports you can see that the number of recorded cases in which the eye symptoms seemed to have a causal connection with a suppurating sphenoidal sinus, and which were proven at the necropsy, are still comparatively rare.

This, no doubt, can be accounted for by the protection of the sphenoidal bone, whose upper and outer walls are much thicker than the anterior, through which the natural opening for discharge occurs.

As you can see upon the coronal section, through the frozen specimen, in which the cavernous sinus is cut through just in front of the optic chiasm, the contained vessels and nerves, going to or returning from the orbit, are protected by quite a thick bony wall from the sphenoidal sinus. Doubtless in rare cases the protection is not so complete, and it is in these cases that a causal relation may exist between a suppurating sphenoid and certain definite ocular symptoms. When extensive necrosis of the body of the sphenoid is present, then the ocular symptoms may be easily accounted for.

While my patient denied a specific history, yet the extensive destruction of bone found at the time of operation, and later at the post-mortem, was strongly suggestive of a syphilitic origin.

In this case with the eye symptoms coming on so suddenly, following the bony necrosis of the sphenoid, I think there can be no question of the causal relation existing between the two.

CASE REPORT.

Mr. E. H., age 32, came on my service at the Cincinnati Hospital, in October, 1906. He was referred by Dr. W. F. Moss, of Maineville, Ohio, for an empyema of the right accessory cavities of the nose, followed by great mental deterioration, with sudden paraly-

sis of the right external rectus muscle and blindness of the right eye. The doctor states that the patient was apparently well up to three months ago and was able to follow his calling, that of a conductor on a traction line.

Status Praesens:

The patient walks with a staggering gait, so that he has to be assisted. His mental condition is such that he can not give a clear history of his case. There is some exophthalmos of the right eye which turns toward the nasal side from complete paralysis of the external rectus muscle. The position of the eye ball is about 5 m. m. below the horizontal line of the other eye. Vision of the right eye nil. Vision of the left eye about normal. Ophthalmoscopic examination showed almost complete atrophy of the optic disc, more marked on the temporal side. The blood vessels, however, were nearly normal in size and number. This is the last expression of a retro-bulbar neuritis, doubtless secondary to an inflammation in the right posterior ethmoidal and sphenoidal cells. Examination of the left eye showed it to be normal.

Dr. D. T. Vail, saw the case in consultation and verified the ophthalmoscopic examination, and considered the condition due to suppuration in the sphenoidal sinus.

The patient complains of great pain at the occiput and at the base of the brain. There is a most offensive discharge from the right nostril, both anterior and posterior. The probe shows extensive necrosis of the posterior ethmoidal cells and the sphenoid sinus. The left side was not affected. Transillumination showed very dark area over the right maxillary antrum with absence of the pupillary reflex. The left maxillary antrum was clear and the pupillary reflex well marked. There was no difference between the reflex in the right and left frontal, both showing clear. Two X-Ray pictures were taken and both corresponded in every particular with the transillumination test.

Under chloroform anaesthesia the right frontal sinus was opened, to be sure no infection was present, since it seemed hardly possible for any sinus to escape, so extensive was the necrosis on the right side. However, the sinus appeared perfectly healthy and the wound was at once closed. The middle turbinate was removed and the necrotic ethmoid cells broken down. The maxillary antrum was entered through the canine fossa. When the antrum wall was exposed it was found to be black from the necrotic condition

present within the antrum. When the antrum was opened a most sickening odor escaped along with pus and necrosed bone. The nasal wall was removed by the Caldwell-Luc method.

The sphenoid could now be inspected and upon attempting to remove the anterior wall it was found that the whole right half of the sphenoid bone was necrosed and movable. With a finger in the pharynx, this loosened condition could be easily made out. Under the guidance of the finger in the pharynx, alligator forceps were passed in through the opening in the canine fossa, and several large pieces of necrotic bone were removed. Others could be felt but I feared to remove them lest the cavernous sinus might be injured. There was present the most extensive bone destruction I had ever encountered and lead me to believe that the disease must have existed longer than three months, as stated by the patient. It was difficult to see how a patient could exist with such an extensive necrotic condition present.

The cavity was packed with iodoform gauze through the opening in the canine fossa. The patient rallied nicely from the operation and on the following morning said he felt much better than he had for a long time, since the pain at the top of the head and back of the neck was gone. He never complained of the pain afterwards.

For several weeks following the operation his mental condition improved. The wound over the right frontal healed by first intention. Three weeks later he succeeded in tearing the wound open, by constantly picking at it, and the frontal sinus became infected. About this time also, the right ear began to discharge. On the fourth week following the operation a persisting and exhausting diarrhoea developed followed by a diffuse and severe bronchitis and the patient died, December the third, five weeks after the operation.

POST-MORTEM NOTES.

Post-mortem by Dr. Frank Hegner, Pathologist to the Cincinnati Hospital. Adult, male, markedly emaciated, phthisical chest, post-mortem rigidity absent. Above internal angular process of the right eye there is a small wound of a previous operation. Foul odor about the head.

Brain—On removing the calvarium and opening the right frontal sinus, a sero-purulent exudate was found in the latter cavity. This

same inflammatory condition was found in the tissues of the orbital fossa, and with increasing severity to complete necrosis in the right anterior and posterior ethmoidal cells. also the right sphenoidal sinus, and the left posterior ethmoidal sinus. To a less degree in the left anterior ethmoidal cells. The process caused necrosis of the floor of the orbit, and extended directly into the antrum, the posterior and lateral walls of which were completely necrosed. Nothing remained of the sphenoidal sinus excepting debris.

Communication between the right middle cerebral fossa and the pharynx was direct. The right middle and internal ear showed sero-purulent inflammatory changes.

Membranes—Dura in the right middle fossa was markedly thickened and very firmly adherent to the sphenoidal fissure and adjacent bone. Over the body of the right sphenoidal bone this thickened dura formed the only barrier between the brain and the necrotic sphenoidal sinus.

Pia and arachnoid showed over the right hemisphere, anteriorly and inferiorly, marked lepto-meningitis. On section no macroscopic lesion was found in either hemisphere.

BIBLIOGRAPHY.

1. Rouge. *Union med.* Paris, 1872.
2. Schaeffer. Chirurg. Erfahrungen in der Rhinol. und Laryngol., Wiesbaden, 1885.
3. *Boston Med. and Surg. Journ.*, Dec., 1903.
4. Nasal Suppurations, 1901, Gruenwald, page 280.
5. *Arch. Ophth. and Otol.*, N. Y., Vol. xxv, 1896.
6. *Journ. Laryngol.*, London, Nov., 1902.
7. *Journ. Laryngol.*, London Dec., 1904.

PAPER:

PURULENT AFFECTION OF THE LABYRINTH CONSECUTIVE TO DISEASE OF THE MIDDLE EAR. PATHOLOGY.

CLARENCE JOHN BLAKE, M. D.

The intimate study of suppurative invasion of the labyrinth is sufficiently modern to give it the zest which attaches to research, and, even in as limited a field as the consideration of the pathologic processes incident to its implication in a suppurative disease of the middle ear, it is necessary to make the starting point from the normal and to include, in a preliminary review, such accepted facts in regard to the structure and relationship of the labyrinth, and such provisions as are accorded it for defense, as may have a bearing upon the changes incident to disease.

The labyrinthine capsule while fairly uniform in itself both as to its intimate structure and its relationship to the surrounding portions of the petrous bone, varies greatly in its juxtaposition to the neighboring tympanic cavity, this variation being due to irregularities in the contour of the tympanum, epitympanum and aditus and the differing degrees in which both the pneumatic spaces and their surrounding diploe encroach upon the base of the petrous pyramid, thus affording readiness of access of pyogenic material from the tympanic spaces to the labyrinthine capsule.

Another variable element which has to be considered, and which is especially important to the study of labyrinth necrosis and sequestrum formation, is the diploetic layer very nearly surrounding the labyrinth in the new born but gradually decreasing by the encroachment of more solid bone until, in the adult, contact suffices to make the labyrinthine delimitation almost impossible, in some places, and representable only by a slit-like line of demarcation in others.

Regarded from the tympanic side, the direction from which the form of attack, under present consideration, is made, the most

vulnerable points, would appear to be the windows, the horizontal semicircular canal and the promontory, the proportionate degrees of vulnerability being perhaps fairly expressed, in the light of present knowledge, in the order in which these parts are named, though a still larger material than that thus far afforded and a more definite record of findings will be necessary to arrive at absolute determination, especially as the review must cover not only the more acutely destructive processes, incident to certain systemic conditions, but the slower, necrotic, abolition of sound tissue incident to prolonged suppuration of the middle ear.

Regarded structurally, in its relationship to a suppurative middle ear, the intact labyrinth presents a capsular environment jeopardized by relationship to the source of invasion, hazarded by anatomical approaches which penetrate its walls, and safeguarded from within mechanically, by the extrusive force of its fluid contents. The jeopardizing relationships are those of the first whorl of the cochlea, in the promontory, the outer vestibular wall and the horizontal semicircular canal, the anatomical approaches are the oval and round windows and the vascular penetrations of the capsule, and the mechanical protection the constant outward pressure and, where penetration of the capsule has been effected by caries, the outflow of the perilymph, effectual only up to the point of general participation of the labyrinthine contents in the suppurative disease.

In the more limited caries the points of attack are those which are most favored by propinquity and by structural weakness while in the more general necrosis the lines of least resistance are those which include, as constant factors, the oval window, on the outer and the porus acusticus internus on the inner vestibular wall and, as a variable factor, the extent of the diploetic remnant enclosing the labyrinthine capsule.

The interstitial granulomatous process which constitutes the basis of a limited caries is especially forwarded by contour conditions which permit accumulation of debris and the correspondingly unhindered activity of pyogenic organisms, while the still debatable question of relationship between the circulation of the periosteal submucosa of the tympanum and that of the intralabyrinthine periosteum, as on the promontory for instance, becomes of less importance in view of the rapid vascular proliferation which takes place when once the interstitial granulomata have become established. The fact that these new growths belong, func-

tionally, to the class of extrusive granulomata, does not lessen their influence, as media for vessel proliferation, in the gradual weakening of the underlying bony structure, or their facilitation of the invasion of such complicated soft tissue combinations as are found about the foot plate of the stapes.

The niche of the oval window, with the stapes in position, affords a favorable opportunity for the progressive development of a limited caries, both because of its contour and of the liability to the blocking, within it, of a pyogenic process favored by the engorgement of its exceedingly vascular soft tissues, while the tissues forming the substructure of the foot plate of the stapes and the propinquity of the nervi duct of the facial and of the ampullary enlargement of the semicircular canal, on the superior posterior niche wall, give ready access to the labyrinth interior when they have become perforated.

An element of defense, under these conditions, but one which can only be theoretically estimated, in default of more intimate clinical observations, is the tendency to outflow of perilymph which continues so long as an opening in the labyrinthine wall persists and the aqueductal communication pertains. In some cases of removal of the stapes, the labyrinth being intact, a steady outflow of perilymph has been observed for several days, gradually decreasing as the rent in the basal membranes closed and ceasing finally only when the repair was complete. In view of this fact it is at least supposable that, with a small opening through the labyrinthine capsule, the product of a limited caries, the tendency to outflow would be measurably protective until a general pyogenic invasion of the labryinth, with engorgement of the membranous structures, and the occurrence of the fibrinous deposits, had cut off the aqueductal supply.

When once the labyrinth has become invaded the spread of the inflammatory process throughout its intimate structures becomes rapid and complete, the interference with the vitalizing blood supply progresses and the negative process of necrosis tends to supplant the active invasive process of caries; the chronologic sequence, in any given case, being dependent both upon local structural and general systemic conditions; in the exanthemata of childhood the whole process, from the primary invasion of the labyrinth to a more or less extensive necrosis, may be very short, while in adults, as the result of a long standing chronic middle ear suppuration

with cholesteatoma, it may extend over years, but however limited in area the true necrosis of the labyrinthine capsule may be, the participatory effect upon the membranous labyrinth is generally one of complete destruction.

That the labyrinth windows afford an especial opportunity for the passage of infection from the middle ear into the labyrinth is evident both from their structure and from the literature describing the operative and post mortem findings in which intralabyrinthine suppuration was accompanied by destructive invasion of the vestibule through the medium of the opening, closed only by soft tissues and the base plate of the stapes, which includes so considerable an area of the outer vestibular wall.

The common clinical evidence of the frequency of limited caries, with redundant extrusive granulomata, in the niche of the oval window in cases of chronic, prolonged, suppurative disease of the middle ear is suggestive of the predeliction of this one of the anatomical pathways to the labyrinth, a predeliction in which the round window shares, though in a lesser degree, its position, the planes of its inclination, and its freedom from superimposed structures, rendering it less liable to attack than if it were placed at the bottom of a deep depression.

The view of Hinsberg, that a considerable swelling of the tympanic mucosa serves to protect the membrane of the round window, is directly controvertable by the fact that such a condition, by retaining pyogenic products, and favoring superficial ulceration, would tend to rob the membrane of the round window of its anatomical advantage.

Unlike the lining of the pneumatices of the mastoid, and of other portions of the tympanum, related thereto and to the tympanopharyngeal tube, the covering of the inner tympanic wall, in the region of the windows and over the promontory, more especially, exhibits the characteristics which constitute the sub-mucosa a periosteum and it is here that the progressive sequences of superficial erosion, hyperaemia, pathologic exudate, the interstitial granulomatous invasion of caries and the secondary destruction of necrosis are most displayed. Friederich says: "The most important evidence elicited in the examinations made by Panse, Habermann, Hänel, Manasse and myself, is that the fenestral niche was filled with granulomata and nascent connective tissue, the stapes being frequently fairly embedded in masses of granulations which,

furthermore, had destroyed the annular ligament and thrust themselves into the vestibule, while not infrequently, in addition to implication of the stapes, its cartilaginous base and the bony rim of the oval window itself were extensively carious, and, (as might be expected from the complexity of the structures at the bottom of the oval niche), the most varied pictures of the passage of the inflammatory process into the labyrinth were presented. The arms of the stapes were either partly destroyed or had disappeared altogether in the enveloping mass of granulations, the base plate of the stapes showed limited areas of caries on the tympanic side, or had been entirely destroyed, remnants only of the carious bone being found amidst the granulations; in one case the whole of the posterior superior niche wall forming the boundary of the nervi duct of the facial had been replaced by granulations."

The extent to which the membrane of the round window participates is only with difficulty ascertainable, the configuration of its niche is much more variable than that of the oval window, especially on the anterior wall where the bony overhang is frequently such as to afford an opportunity for the accumulation of septic products.

It is this question of the accumulation of septic products which has an important bearing upon the invasion of the labyrinth, directly through its bony wall. Through the operation of the pathologic sequence already mentioned the two next most exposed portions, in their relation to the primary field of disease, are the promontory and the horizontal semi-circle canal, but while the promontory is the most exposed it is also, from its position, the more amenable to drainage, affording a striking contrast to the semi-circle canal which, often closely approximated to, if not abutting upon, the recessus epitympanicus, is in close relationship to a frequent seat of septic retention and granulomatous activity. It is but rarely, in the course of a prolonged suppuration in the middle ear, that the labyrinth is invaded by one channel alone and this is probably particularly true in those cases in which accumulations of desquamated epithelium have been either the covers for septic attack, or, by their pressure, have contributed to lower the vitality of the attacked area, and the frequency of this form of complication of a protracted middle ear suppuration, in the epitympanum, probably contributes to the greater frequency of fistulous penetration of the semi-circular canal.

There is still considerable difference of opinion as to the relative degree in which the semi-circular canal is the seat of primary invasive lesion, the discovery of a fistulous opening, through the medium of an exenteration, in default of substantiation of the integrity of other channels, being only presumptive evidence, since, wherever invaded, the labyrinth interior so rapidly participates, throughout, in the suppurative process; though Politzer cites cases in which the intralabyrinthine changes were limited to the region of the windows, and other observers report instances of limitation within the primarily invaded semi-circular canal. The prognosis for a limitation of the suppurative process in the horizontal semi-circular canal is favored by the readiness with which the limited area is blocked off by the results of the interior inflammation precedent to the actual fistulous opening; the limitation evolved by the inflammatory process in the vestibule is, on the contrary, exceedingly rare, as shown by the observations of Breiger, though Politzer has observed cases in which the changes in the vestibule were accompanied by only moderate alterations in the cochlea. In contradistinction to the opinion of Jansen, Politzer also regards empyaema of the intradural prolongation of the acqueductus vestibuli as very rare, he having but once found suppurative products in the ductus endolymphaticus and once in the ductus perilymphaticus.

It is in the suppurative implication of the cochlea that the prognosis for limitation is least favorable, the various anatomical channels for the passage of septic material, the readiness in inflammatory participation of the diploetic structure of the modiolus, and the thinness of the multifariously perforated terminal wall of the porous acusticus internus, affording ready passage along the channel of the auditory nerve.

While even a cursory review of the augmenting literature of this subject shows the question of transference of a suppurative process from the middle ear to the labyrinth, to fall, with exception of certain mooted points, within simple lines, the question of suppuration within the labyrinth itself, of periosteal involvement, of attacking caries, necrotic delimitation and of the structural paths through which these pathologic changes are effected, is beyond the bounds, without undue lengthening, of the present communication, and a worthy subject for distinct consideration in another symposium.

Viewed from the standpoint of the middle ear, wherever the attack may be made and whatever the degree of distortion or of destruction, the pathological sequence is the same, from the superficial layer of the soft tissue lining of the tympanum inward, into and through the underlying bone; irritation, hyperaemia, congestion, normal exudate, maceration, erosion, pathologic exudate, accumulation of detritus and formation of extrusive granulomata; separation of the periosteum, penetration of the outer cortical layer and suppurative extension through the haversian canals, the destruction of the bone being further favored by the invasive interstitial granulomata, with their newly created blood vessels, and their blocking of the normal blood supply, producing a tissue change which is both a caries and a necrosis intimately combined. With penetration of the labyrinth capsule there follows separation of the endosteum and, with its penetration, and the sequent interior involvement, the invasive cycle is complete.

One element of the middle ear secretion which needs to be taken into consideration for the part it may play, in what may be called the mechanism of septic invasion of the labyrinth, is the mucin, that albuminoid substance which, in excess, imparts the peculiar ropy character to the tympanic secretion and so favors aggregations of septic detritus and provides a nidus for bacterial activity; it is precisely in such depressions as that of the fenestral niche that such an element may become effective in favoring a pathologic process.

Not only does the presence of mucin accord with bacterial development but it may be itself a product of bacterial agency. Rettger,[1] in recent investigations shows that the production of mucin or a mucin-like substance is not limited to one or a few species of bacteria.

There is considerable evidence that the process of mucin production, by bacteria, is not a direct one; there is, presumably, formed an intermediate body, a pseudo-mucin, to the presence of which the younger cultures owe their marked slimy consistency; as development goes on this peculiar substance becomes gradually transformed into the more soluble type of mucin, hence, as a rule, the older the culture the larger the amount of mucin contained in it; the rapidity with which the pseudo-mucin is transformed into the final product varying, to large extent, with the bacterial species.

A brief review of the systemic conditions favoring labyrinthine septicaemia shows that the exanthemata of childhood, especially scarlet fever and measles, are acutely important, while of the more chronic disorders, tuberculosis, diabetes and syphilis are influential in frequency in the order given, and it is in the presence of these diseases that the necrosis of larger area and the major sequestræ more commonly occur.

Klug[2] reports six cases in which there were nine labryinth suppurations, five of the cases demonstrating the destructive influence of scarlet fever; in one case, in which the etiology was obscure, the predominating mechanical influence was cholesteatomatous.

In the first case a portion of the cochlea was involved; in the second there was a fistulous opening into the horizontal semi-circular canal and pus issued on opening the vestibule; in the third the vestibule and cochlea were filled with pus and there was a small fistulous opening in the annular ligament of the stapes; in the fourth the sequestrum, removed from the left side, included the cochlea and a portion of the vestibule and of the semi-circular canals, while on the right side, in the same case, there was merely a porous necrotic mass which included the major portion of the labyrinth; in the fifth case the sequestrum, from the right side, included the three semi-circular canals and a portion of the cochlea and that from the left side, two of the semi-circular canals and one whorl and a half of the cochlea; in the sixth case the sequestrum, from the left side, included a portion of the vestibule and one and one-half whorls of the cochlea, the remainder of the sequestrum being an indefinable mass of disorganization and, on the right side, there was a fistulous opening into the horizontal semi-circular canal, a similar opening through the promontory and a corresponding suppurative implication of the canal and of the cochlea.

The pathological changes in the labyrinth capsule, and its surrounding diploe, in uncomplicated chronic suppurative middle ear disease, are stated by Politzer[3] to consist in no more than abundant cell agglomerations in the bone cavities and in hyperaemia while, on the contrary, in tuberculosis and corresponding cachexiae and in sepsis, or with the cholesteatomatous complication of prolonged suppurative middle ear disease, more or less extensive defects in the bony capsule are determinable.

The descriptions, by various observers, of these changes, and the multiform combinations of different stages of the pathologic sequence which they present are strikingly illustrative of the variations in a process which may be either rapidly completed or may take years for its consumption, according to the relative balance between resistance capacity and the conditions attendant upon the invasion.

The convexity of the semi-circular canal and the promontory have been found to be partially carious, wholly necrotic or exhibiting numerous small openings filled with penetrating granulomata, separated from the labyrinth interior only by the elevated endosteum, or, where this also had participated in the destruction, with an established communication between the septic middle ear and the perilymphatic space. The windows exhibited the greatest variety of changes, in some instances the stapes and its annular ligament remained intact, in others the ligament was variously perforated or entirely destroyed, the cruræ were partly or wholly wanting, the base plate either carious in several places, on its outer surface, and either displaced or represented only by a small remnant of infected bone embedded in the swollen mucosa or in the mass of granulomata which had attained free access to the vestibule.

The membrane of the round window was enclosed in the swollen mucosa, in some instances greatly thickened in others perforated, or entirely destroyed, and the lumen of the window filled with penetrating granulomata.

The pathological changes within the labyrinth itself have been shown to be equally various, from a circumscribed area of exudate on the inner surface of the foot plate of the stapes and on the inferior wall of the vestibule, or organized connective tissue, in the same location, the other portions of the vestibule remaining intact; to a partial or complete filling of the vestibule with pus, hemorrhagic exudate or highly vascular connective tissue in communication with the granulomatous tympanic mucosa through the medium of the windows or of fistulous openings through the promontory or other portions of the bony capsule. Similar changes were found also in the cochlea and in the semi-circular canals, in some instances the pus, entering the basal portion of the cochlea, penetrated the tractus spiralis foramenulentus or made its way along the channels of the nerve fibrils or through created defects

into the porous acusticus internus; in some instances the modiolus was mainly intact, in others almost completely destroyed and, presumably, the point of departure for septic incentive to multiple caries of the outlying labyrinth capsule.

In the majority of cases of implication of the cochlea the organ of Corti was destroyed or at least so changed that the details of its structure were undeterminable.

One other frequent accompaniment of the septic invasion of the labyrinth should not pass unmentioned and that is the paralysis of the facial, incident to the penetration of its bony canal at some point in the relationship of its course to the septic middle ear or labyrinth. The proportion of facial paralysis, either partial or complete, as given by the two observers covering the largest amount of material in this respect, Gerber[4] and Bezold[5] varies from 77 per cent to 83 per cent, the most vulnerable points of the nervi duct, to access from the middle ear, being the region above the oval window and the superior portion of the posterior tympanic wall.

The proportion of cases of labyrinth implication in suppurative disease of the middle ear, as given by various observers, ranges from 0.77 per cent to 1.2 per cent and the estimate of Friederich[6] that there is one labyrinth implication in every hundred cases of suppurative middle ear disease presents, therefore, a fair general average of the results of present observations.

In view of the possibility of an increase of this percentage as this vital subject comes to be more generally, and more intimately studied, and in view also of the evidence, already accumulated, of the extent of destruction within, and including, the labyrinth capsule, when once septically invaded, the questions of symptomatic indication and of defined surgical procedure become of the greatest importance.

BIBLIOGRAPHY.

1. L. F. Rettger. Mucin as a Bacterial Product. *Journ. of Med. Research,* Vol. X, p. 101.
2. Klug. Les suppurations der labryinthe. *Ann. d. mal. de l'oreille du larynx, etc.,* Feb. 1906.
3. Politzer. Labyrinth Befunde bei chronischen Mittelohreiterungen, *Archiv. f. Ohrenh.,* Vol. 65, Nos. 3 and 4.
4. Gerber. *Archiv. f. Ohrenh.,* Vol. LX.
5. Bezold. *Ztschr. f. Ohrenh.,* Vol. XVI.
6. Friederich. Die Eiterungen des Ohrenlabyrinths.

PAPER:

THE SYMPTOMATOLOGY AND DIAGNOSIS OF LABYRINTHITIS, CONSECUTIVE TO PURULENT OTITIS MEDIA.

H. O. REIK, M. D.

The magnitude of the task I have undertaken fairly overwhelms me. When our secretary, with sweet words and characteristically persuasive manner, invited me to take part in this symposium I readily, yea, even gratefully, accepted for I was proud of the privilege of appearing in company with Drs. Blake and Richards. For the time being I failed entirely to appreciate the fact that, with the honor, he had conferred upon me a heavy obligation. It appears to me now that the topic assigned to me is by long odds the most difficult portion of the subject: the pathology of purulent invasion of the labyrinth is fairly well worked out, or the missing links may be supplied by hypothetical reasoning, and, treatment of the disease consists in the application of established surgical principles.

The diagnosis of a disease is the first, if not the most, important step in its consideration and the title of this paper would imply that the author should present such a clearcut set of rules as would, upon application to a concrete example, enable one to determine with reasonable accuracy whether or not labyrinthine suppuration existed. Before you condemn me for failure to measure up to those expectations, I beg you to remember that much of the blame should fall upon Dr. Harris for having shown such lack of wisdom in selecting for this role the least capable of his performers. That I shall fail is inevitable, not only because my own experience with the disease has been, necessarily, limited, but because in the whole literature of the subject there is not, as yet, a sufficient number of accurate clinical records to permit the construction of a sharply defined clinical picture. No one man has observed any great number of cases and Gerber, in 1906, was able to find only 90 satis-

factorily recorded cases. You will pardon me, therefore, for supplementing my limited personal knowledge of the disease by frequent reference to the published experiences of others.

The publications which I have found most helpful in this study, and the reading of which will well repay anyone interested in the subject, are: "Sur le diagnostic et le traitment des suppurations du labyrinth." Von Stein., Ann. d. mal. de l' orielle etc., 1905, XXXI p. 30; "Labyrinthinekrosa." Gerber., Arch. f. Ohrlk. 1904, LX, p. 32; and, that most excellent monograph, comprising a resume of the entire subject of labyrinth suppuration, of Friedrich's "Die Eiterungen Krankheiten des Ohrlabyrinths." From these I have abstracted freely.

Bezold estimated that there is but one case of labyrinth necrosis in 500 cases of chronic purulent otitis media; a surprisingly small percentage when one considers the large number of existing cases of chronic middle ear suppuration which are absolutely neglected for long periods of time. A possible explanation of the small number of cases seen lies in the fact that the disease occurs most commonly in the first decade of life, and we all know how frequently the ear is overlooked in children suffering from grave pyemic conditions. Of the 90 recorded cases, according to Gerber, 37 occurred in children under 10 years of age; his table being arranged as follows:

1 to 10 years old	37 cases
11 to 20 years old	15 cases
21 to 30 years old	16 cases
31 to 40 years old	7 cases
41 to 50 years old	8 cases
51 to 60 years old	5 cases
61 to 70 years old	2 cases

A further explanation is found in the mistakes of diagnosis, particularly because of the difficulty of distinguishing between labyrinthitis and cerebellar abscess. It is fair to presume that many of the fatal cases of ear disease in which the diagnosis of meningitis or cerebellar abscess was made, and upon which no autopsy was performed, had really passed through the stage of labyrinthitis or were instances of abscess localized in the labyrinth rather than in the cerebellum.

Chronic suppurative otitis is almost always the character of the middle ear disease preceding purulency of the labyrinth; very

rarely does the inner ear become involved during the acute stage of otitis. There is no fixed period of chronicity, however, necessary to allow the deeper invasion and the time intervening, in the reported cases, varies from 6 weeks to 31 years. One case has come under my own observation in which the otorrhoea had existed but 5 months and the history indicated that the first evidence of labyrinthine trouble, a severe attack of vertigo, with tinnitus, nausea and deafness, appeared when the middle ear disease was only two months old.

As predisposing factors, some constitutional dyscrasia or the exanthematous fevers are generally present; tuberculosis and diabetes, scarlet fever and measles, being the most frequently noted. There is no difference in vulnerability as regards sex, nor between the right and left ears. The extent of destruction in the 90 studied cases shows:

Necrosis of entire petrous portion of temporal bone	7 times
Complete labyrinth necrosis	17 times
Complete cochlear necrosis	19 times
Partial cochlear necrosis	26 times
Necrosis of parts of cochlea and labyrinth	10 times
Labyrinth necrosis without reference to cochlea	5 times
Necrosis of semi-circular canals alone	2 times
Extent of process not known	4 times

It will thus be noticed that the cochlea was concerned in 79 out of 80 cases and that the lesion was limited to the cochlea in 50 per cent of all the cases. If, in addition to this, we consider the further fact that it is the first convolution of the cochlea which is most frequently affected, we have a confirmation of the clinical observation that the round window is the weakest point on the inner tympanic wall and the most frequent route of labyrinthine invasion.

A detailed study of the symptomatology of purulent otitis interna, (suppurative labyrinthitis), may be facilitated by treating under separate heads the general and special symptoms, and the latter will have to be considered as subjective and objective phenomena.

GENERAL SYMPTOMS.

1. *Fever.* The febrile condition in this affection shows nothing characteristic. Instances of the most extensive and diffuse de-

struction of the labyrinth are recorded in which the temperature was never a full degree above normal at any time during the illness. Subnormal temperatures have been noted not infrequently and in at least one case continued so until within a few days of death. With the previously long existing suppuration in the tympanic cavity, to which the system has become more or less adjusted, and the slowly progressing attack upon the structures of the internal ear, we might expect that the labyrinthine disease would advance without material febrile disturbance. That is what usually happens. The attention of the otologist is attracted to the spread of purulent otitis to the inner ear not, as in other complications, by temperature changes, but by distinctly aural symptoms, and, when in the course of labyrinthitis a marked fever arises it is an indication of the appearance of intra-cranial complications, usually lepto-meningitis.

2. *Headache.* This is a common accompaniment of all forms of temporal bone inflammation, and especially of their cerebral complications, so that it can not be regarded as of much importance in the recognition of labyrinth disease. Some degree of earache or headache, occasionally acute pain but more often a dull sense of soreness in the head, is present in most cases. There is a general agreement that very violent pains in the ear and head precede sequestration of a necrotic labyrinth; the pain disappearing after expulsion of the sequestrum.

3. *Nausea and Vomiting.* Like the above named symptoms, nausea and vomiting, considered alone, are of very little diagnostic value. It is only when occurring in connection with the other symptoms of Ménière's group that any importance attaches to their consideration. Bezold long ago pointed out, however, that giddiness, nausea and vomiting occurred in the early stages of labyrinth necrosis and that their appearance in a case of chronic suppurative otitis media is always suggestive of beginning labyrinthine involvement. The primary attack of vertigo may be, and frequently is, very violent, necessitating the patient's confinement to bed. In one of my own cases, the patient had two such attacks, each of which kept her in bed for more than a week. These, as well as the other evidences of disturbed equilibrium, tend to diminish in intensity, or to disappear, as the disease progresses; being dependent upon irritation of the vestibular apparatus, they are not repeated after the disease has destroyed the nerve terminals

in the ampullæ. The possibility of giddiness and nausea being produced by strictly intra-tympanic disease must not be overlooked. We have all observed cases of this sort.

SPECIAL SYMPTOMS AND THEIR INVESTIGATION.

1. *Tinnitus Aurium.* Complaints of subjective auditory sensations are surprisingly rare in disease of the labyrinth consecutive to purulent otitis media, and when occurring seem to be of temporary duration only. The omission of reference to tinnitus in the reported cases is particularly striking in view of the fact that this symptom is so constant a factor in other diseases of the labyrinth. Bezold found it recorded in but 3 of 41 published cases and did not observe it at all in 5 cases under his own care. Buzzing, humming and ticking are the sounds generally described, but, in a few cases, there have been extremely loud noises—like the puffing of a locomotive. Hinsberg suggests that the acoustic phenomena of irritation may not have been absent in all the cases in which they were not mentioned, but Friedrich's explanation seems the more probable—that "with the gradual development of the clinical symptoms of labyrinth suppuration, ear noises do appear in the beginning as irritation symptoms which, later on, disappear with the destruction of the nervous apparatus."

2. *Impaired Hearing.* Recalling the fact that the cochlea is the part of the internal ear primarily affected in the majority of cases, and that it is injured to some degree in 90% of all cases, we are prepared to expect that deafness should be one of the most regular symptoms of labyrinth suppuration. Gerber says that in 67 of the cases tabulated, in which the defect of hearing was recorded, there was complete deafness in 43 and some remnant of hearing only in 22. Accurate proof of the amount of hearing retained is not easily obtainable. The objective tests of the examiner are to an extent controlled by the subjective sense of the patient and the way is open for inaccuracy and false deductions. Furthermore, even with the greatest care, and when dealing with a helpful patient, it is extremely difficult to make sure of occlusion of the other ear. In fact, it is practically impossible to completely shut out the good ear. One must, therefore, be very cautious about accepting evidence of remaining hearing in a case where other symptoms point to extensive destruction of the sound perceiving organ. Especially is this true of the employment of the voice, as a testing medium, and of the higher tuning forks.

The functional defects characteristic of labyrinthine disease are: Impaired bone conduction, as tested by the tuning fork; reduction of the range of audition, the loss being perceptible from both the lower and upper ends of the scale; diminished air conduction, so that the Rinne test proves negative; and, lateralization of the Weber test towards the unaffected ear. Total destruction of the cochlea means total deafness. Partial destruction of the cochlea may leave still some hearing power for the middle register, but even here the duration of both air conduction and bone conduction will be reduced. One can imagine invasion of the internal ear through the semi-circular canals without material impairment of hearing for the higher notes, because the perceptive apparatus has not been seriously attacked, but autopsy examinations prove that this condition is very uncommon, at least in the graver cases that have come under observation.

3. *Coördination Disturbances.* There is apparently some difference of opinion as to the frequency of these symptoms, giddiness, nystagmus, vertigo and nausea; Bezold seems to believe that to some extent they accompany most cases, while Gradenigo holds that they are absent in many cases. The latter finds such symptoms unrecorded in just about the same percentage of cases as are known to have the lesion confined to the cochlea, and suggests the probable correspondence of the conditions. That is, that when necrosis is limited to the cochlear portion of the internal ear there will be no disturbance of coördination. On the other hand, it must be noted in this connection, that in some instances of undoubted semi-circular canal affections there has been complete absence of any disturbance of equilibrium.

Friedrich endorses Bezold's view and considers equilibrium disturbances to be, next to impaired hearing, the most constant symptom of labyrinth disease. He includes among such disturbances: giddiness, standing or walking, with closed or open eyes; vertigo, even in the position of rest; horizontal nystagmus, appearing especially when glancing towards the healthy side; and attacks of nausea and vomiting. He believes that they are but temporary phenomena and never assume a permanent character. That there may be cases which, because of the very slow advance of the carious process, never show these symptoms, he admits. Although Bezold is accredited with the opinion expressed above, his statistical report of 46 cases of labyrinth suppuration includes only 12

cases in which attacks of giddiness were reported. Friedrich supports his contention by reference to the investigations of other workers, as well as his own clinical experience. Thus, Jansen found giddiness present in 72% of his cases of carious and traumatic lesions of the labyrinth; Lucae, in 60%; Hinsberg, in 86%; and Friedrich, himself, in his 27 cases of labyrinth suppuration, found that giddiness was present 16 times, absent 8 times, and the records were missing for 3 cases.

The subjective vertigo complained of is the sensation of falling towards the healthy side or the turning about of neighboring objects. When walking, there is giddiness and uncertainty of movement. When lying down, giddiness and the fear of falling out of bed.

To test the sense of equilibrium, Von Stein has offered us two special devices, the movable inclined plane and the revolving chair, both of which instruments are designed for the purpose of determining accurately the angle of relative position or the degree of rotation required to produce disturbances of sensation. In a very recent article, after relating an interesting case, he says, however, "total or partial necrosis of the labyrinth is always accompanied by troubles of coördination which are most marked during the jump with the eyes closed. To provoke these symptoms, and bring about a diagnosis in this way, one has no need for special apparatus. All that is necessary is to make the patient walk and jump with the eyes closed. But, to search out a complete diagnosis, and to approximate in figures the degree of these troubles, one should use the *goniometre* and centrifugation, without which it is impossible to make a diagnosis in the initial period." From this it would seem that he has come to the same conclusion as all others who have tried the inclined plane test, namely, that it affords very little assistance in making a diagnosis. The goniometre, which provides for rotary motion and sudden stoppage, is not only a scientific application of this simple test, which can be made with any revolving chair, but affords a means of measuring such rotations, and the effects produced, that may come to be helpful in determining the existence of a lesion in its early stage, or of locating it in the bony capsule, perilymphatic space or membranous capsule.

At the present time, then, our tests of equilibrium are about as follows:

a. Standing, with open and then with closed eyes, to note unsteadiness of position.

b. Walking, with open and then with closed eyes, noticing a tendency to ataxic gait or divergence from a straight line; the tendency to walk in a curve, generally.

c. Jumping, feet and knees in apposition, and eyes closed, to ascertain any tendency to fall toward the affected side.

d. Turning the patient around and bringing the body to sudden stoppage. This should be done first toward the sound side and then toward the affected side to note any complaint of giddiness, this sensation being most likely to be produced with rotation to the diseased side.

One other test for muscular incoördination is that of eliciting nystagmus. The patient is never conscious of nystagmus, and, while it has been observed in a large percentage of the reported cases of labyrinth disease, the physician has to search for it. If the patient be directed to turn the eyes strongly toward the sound side and then upwards, the oscillating movement may be seen, if at all, during the time the eyes are held in such position; the nystagmus in these cases is always of the horizontal, oscillating type, never rotary.

4. *Facial Paralysis.* Bezold looked upon facial paralysis occurring in connection with long-standing suppurative otitis media as a reasonably sure sign of extension of the latter disease to the labyrinth. Friedrich takes issue with him on this point, for in his 27 cases of labyrinthine suppuration facial paralysis was present in only 3, at the time they came under observation. The symptom is of diagnostic value only when coexisting with labyrinthine deafness and disturbances of coördination.

5. *Intratympanic Appearances.* Consideration of the tympanic pathologic alterations is not properly a part of this symposium, but it would be manifestly improper to omit all reference to the disease precedent to and causative of that which we are discussing. It will probably be sufficient to say, that, to complete our symptomatology, as regards gross pathological changes in the tympanum, very little information of diagnostic importance can be obtained by their inspection, and that it is, usually, only during an operation that we can inspect the inner tympanic wall sufficiently well to locate any carious lesion. Pus exuding from the oval window or from the promontory, particularly if it rapidly reappears

after being wiped away, is positively diagnostic of labyrinthine invasion, and the appearance of granulation tissue springing from the inner wall, with roughened bone underneath, is characteristic of necrosis of the labyrinth capsule.

DIAGNOSIS.

A positive diagnosis from the preoperative study of our cases can seldom be made. In the majority of instances treatment must be instituted upon the basis of a probable lesion, the proof of whose existence depends upon an exploratory operation. Even when the internal wall of the tympanum is exposed, it will require the most careful search to find the portal of entry or exit of inflammatory products. Granulations protruding from the oval or round windows may be recognized, but carious fistulæ are not so readily discovered; they must be sought with a fine probe. When they cannot be found, it may occasionally be observed that the semi-circular canal wall presents a bluish or brownish discoloration, due to the appearance of granulations or blood-clot as seen through the thin bony covering.

The natural sequel of untreated purulent otitis interna is death by leptomeningitis or encephalitis. Consequently, as most of the observed cases are the result of neglect, we have to make a differential diagnosis between labyrinthitis and the intracranial complications of otitis media. In leptomeningitis, we have to guide us, the characteristic persistent high fever and the changed appearances of the cerebro-spinal fluid, as examined after lumbar puncture. In cerebral abscess, without localizing signs, low temperature, subnormal at times, accompanied by slow pulse, is perhaps the most reliable symptom. Cerebellar abscesses are still more confusing in that headache, vertigo, vomiting and coördination disturbances are the most prominent symptoms.

One is strongly tempted, Mr. President, to take the symptoms enumerated above and try to draw some distinct picture which he could call a clinical entity and to it attach the name of—suppurative otitis interna, or, purulent labyrinthitis. I shall deny myself that joy. The whole subject is, as yet, in too chaotic a state. And, if it were possible, I do not know that it would be wise to draw such a picture at the present moment. David Grayson, in one of his interesting stories on "Contentment," recently described this instinctive desire for completeness in the following

words: "No conclusion ever quite satisfied me. I always knew there was something yet remaining to be told. The only safe conclusion we can reach is this: Life changes. How strange it is, then, that we should be content to take such small parts of it as we can grasp, and to say, "this is the true explanation." By such devices we seek to bring infinite existence within our finite egoistic grasp. We solidify and define where solidification means loss of interest." Anything that might tend to check interest in this problem at the present time would be fatal to our study of one of the most obscure and dangerous complications of purulent otitis media.

PAPER:

SURGERY OF THE LABYRINTH.

JOHN D. RICHARDS, M. D.

The following paper is based upon 11 cases of labyrinthine suppuration and necrosis.* Of these, four occurred in the course of acute or subacute mastoiditis; seven, in the chronic suppuration. In *four,* neither symptoms were present, nor was there anything in the previous histories suggesting inner ear involvement; the labyrinthine invasion was discovered at the time of the mastoid operation.

In *three,* no symptoms were present at the time of operation, though the previous history in each pointed to labyrinthine disturbance.

In *four,* decided symptoms were present at the time of operation.

In *three* of the eleven cases infective sinus thrombosis complicated the labyrinthine disease. In two of these the jugulars were thrombosed.

This relative frequency of sinus involvement is of surgical interest when we consider the vascular connection between the veins of the vestibule, the semicircular canals and the lateral sinus, and those of the cochlear and the inferior petrosal.

* The writer has since operated upon a twelfth case in which a cholesteatomatous mass invaded the internal ear through the oval window, forming a cast of a portion of the labyrinth.

Three of the eleven cases died. The first death was from meningitis resulting from operative interference. The second death occurred in one of the sinus thrombosis cases and was due to meningitis resulting from operative interference. Though the sinus was occupied by an infected clot, the thrombosis was not an important factor in the patient's death, as the post-mortem revealed the pathway of infection to be from the labyrinth along the course of the auditory nerve. The third death occurred in one of the sinus thrombosis cases and was due solely to the infective thrombosis. In this instance the jugular was occluded by an infected purulent clot which extended below the clavicle into the deep thoracic portion of the vein, it being impossible to get below the cardiac limit of the clot. In this instance the labyrinth was in a perfectly healthy condition and had been so for a period of over ten days before the development of the sinus symptoms. The first two deaths resulted from improper technique and the specific errors —for the sake of emphasis—will be referred to in their respective places. I regard these deaths as sacrifices which could now be avoided.

The labyrinthine capsule showed the following lesions:

In five, the only visible lesion was a loss of the prominence of the horizontal semicircular canal.

In one, the oval window was the seat of fistula through which granulations protruded. The vestibule, the semicircular canals, and the lower half of the first cochleal whorl were involved.

In one the prominence of the horizontal canal had disappeared. The oval window was perforated and filled with granulations; a second perforation was present through the centre of the promontory through which polypoid granulations sprouted and pus oozed. The canal system was filled with granulations, the vestibule and cochlea with granulations and pus.

In three, the horizontal semicircular canal had disappeared and the oval window was the seat of fistula and filled with granulations. The canal system, the vestibule and the cochlea were involved in each.

In one, there was a perforation just above the horizontal semicircular canal in the region of the solid angle. The inner vestibular wall was also the seat of a fistula which communicated with a large epidural cerebellar abscess in the vicinity of the internal auditory meatus. The entire labyrinth was involved in the purulent process,

which had also destroyed the greater part of the petrous pyramid, necessitating its removal.

Of the 11 cases, therefore, the horizontal semicircular canal, either alone or in combination with other lesions of the capsule, was involved in nine.

The oval window was perforated in five; the promontory, or first cochleal whorl, in one.

The labyrinthine capsule in the solid angle of the semicircular canals in one.

The inner vestibular wall was perforated once.

In all, there were seventeen perforations in the eleven cases,—sixteen through the outer labyrinthine wall, but one through the inner. This is of surgical importance, and one of two conclusions is evident —either in the great majority of cases the labyrinth is invaded by direct extension through the capsule from without, or else the outer wall of the labyrinthine capsule is exceedingly more vulnerable than the inner. Both are true.

The cases show conclusively that the fistulous openings in the external capsule do not always represent avenues of invasion from without inward, but avenues of escape through which pus is successfully discharged from the labyrinth into the middle ear.

In every instance in which the vestibule has been involved the canal system and the cochlea—either in whole or in part—have been involved also.

The first question which from a surgical standpoint we are called upon to decide—and it is one with which we are frequently confronted—is the following: If after the diagnosis of labyrinthine suppuration is made we operate and find in the outer capsule of the labyrinth no fistula, shall we explore it upon the symptoms, fork tests, etc?

To take a correct surgical attitude with reference to this important point, we will consider first, briefly, the diagnostic value of certain chief symptoms and tests; second, the dangers which attend the exploration of the labyrinth; and third, the dangers which attend the non-exploration of the labyrinth. I trust that a brief reference to the surgical value of the first will not be misinterpreted, in the present paper which is concerned chiefly with the treatment of labyrinthine suppuration.

1st. In the vertigo, vomiting, nystagmus and disturbances of equilibrium of labyrinthine origin, there is nothing characteristic.

2d. Even if we can exclude cerebral and cerebellar disease and can fix these symptoms definitely upon the labyrinth, they are not indicative of actual invasion of the labyrinth; they merely signify disturbance of labyrinthine function; but this occurs with comparative frequency from causes entirely extra-labyrinthine: from polypi, cholesteatoma, pus in the middle ear under pressure, etc.

3d. The mere fact that these symptoms are associated with a suppurative process is of little diagnostic value, as the extra-labyrinthine causes which produce these symptoms by transmitted pressure are, as a rule, associated with a suppurative process also.

4th. Cases occur, presenting all the above symptoms and in addition fork tests, which, according to orthodox interpretation, point to labyrinthine involvement which when operated upon show no evidence of labyrinthine involvement in the outer capsule, and which completely recover without having the labyrinth explored; these cases are not uncommon.

5th. In my own cases the forks have been of doubtful value in differentiating middle ear from labyrinthine lesions.

Briefly: seven of the eleven cases lateralized to the side of labyrinthine lesion. In six bone conduction was not shortened in duration. Of seven in which the greater part of the entire labyrinth was removed, four not only lateralized to the side of the labyrinthine lesion and bone conduction was not shortened in duration, but by air conduction forks were heard throughout a considerable range of the scale.

6th. In every case in which I have seen the labyrinth encroached upon by either operation or disease, the organ has been rendered valueless for practical purposes of hearing. I refer also to several cases of acute mastoiditis in which I have seen the horizontal semicircular canal injured by accident, and which cases I have had the opportunity to examine.

7th. To expose the interior of a normal labyrinth to an infected cavity is highly dangerous to life. What the mortality is under these conditions, I do not know, as I am unacquainted with the literature of this subject; but from my own experience, I realize that the opening of a diseased labyrinth is a serious thing, and I am confident that it is far more dangerous to open a normal labyrinth in the presence of an infected cavity than a diseased one; for in the latter the pathways along which infection may travel to the intra-

cranial cavity and sinuses have had the opportunity to become sealed through inflammatory processes.

8th. If we do not explore the labyrinth under the conditions mentioned, we may overlook certain cases of labyrinthine suppuration—for my own cases show conclusively that the labyrinth is not always invaded directly from without by encroachment upon the capsule, but that it may be infected by septic particles being transmitted to its interior along the avenue of the vessels, the outer capsule showing no perforation whatever, i. e., by a process of local metastasis. The pathological process is analogous to that in which infection of the sigmoid sinus occurs from the antrum along the avenue of the communicating vessels, the intervening mastoid structure showing no apparent involvement.

Should we overlook such a case, we court two dangers:

1. Intracranial infection taking place along the communicating avenues with the labyrinth.

2. That the inner wall of the labyrinth will perforate before its outer wall.

With reference to the first: of the eleven cases mentioned, three had infective sinus thrombosis. This relative frequency throws some suspicion upon the labyrinth as a causative factor, for this proportion of sinus infections is entirely too high for the ordinary run of suppurative cases either acute or chronic.

That the infected labyrinth was responsible for one of the infected sinuses is probable, inasmuch as the symptoms of sinus involvement appeared six weeks subsequent to the cessation of aural discharge. This is suggestive of the role played by the communicating avenues from the labyrinth as infection carriers to the intracranial cavity, and the danger of overlooking such a labyrinthine suppuration.

The frequency with which the cerebellar fossa is found post mortem to be the site of greatest pathological activity in cases of purulent lepto-meningitis, originating from suppurative disease of the petrous pyramid is also suggestive of the important part played by the labyrinth as a distributing center of infection to the intracranial cavity.

With reference to the second—That the inner labyrinthine wall will perforate before the outer, is not borne out by clinical fact, nor, would we suppose it on anatomical grounds.

Of the seventeen perforations in the labyrinthine capsule in the eleven cases, sixteen were through the outer wall, but one through the inner wall. This ratio of sixteen to one is, of course, misleading, inasmuch as the majority of labyrinthine infections occur by direct extension through the capsule from without, under which circumstances the inner wall of the labyrinth is not subjected to a comparative test of vulnerability.

Considering, however, the doubtful value of symptoms as indicative of actual invasion of the labyrinth, the difficulty of eliminating cerebellar disease, the unreliability of tuning forks in differentiating in this class of cases middle ear from labyrinthine lesions, the practical certainty that we will destroy the organ for the purpose of useful hearing, the actual danger to life should we commit the error of opening a normal labyrinth to an infected cavity, the certainty that the outer labyrinthine wall will in the great majority of cases perforate before the inner wall—the correct surgical attitude is, not to enter the labyrinth upon symptoms, etc., alone at the primary operation unless there is direct evidence that the labyrinth is involved. While I can imagine a combination of symptoms and circumstances which would cause me to open the labyrinth at the primary operation, even in the absence of recognizable labyrinthine invasion, I have as yet met no case which presented such features, and it is to be sincerely hoped that the labyrinth will not suffer the indignity of reckless exploration and that it will not be entered without a due consideration of the possible results to life.

What route shall we adopt?

The following facts have bearing upon the selection of the route by which we enter the labyrinth. From a surgical standpoint the cases may be divided into two classes.

Of the first class, the first five cases are illustrative, in which a loss of the horizontal semicircular canal represented the only lesion in the capsule. These cases represent caries of the labyrinthine capsule in which as the extension proceeds from without inward by a process of erosion, the limit to which the labyrinth is surgically involved is, as a rule, represented by the carious area. In such cases we merely remove the carious area and follow it to its limit, and the route which we select depends upon the site of the lesion, which, as a rule, is in the horizontal semicircular canal. The reasons for the frequency with which the horizontal canal are involved are evident.

1st. The external semicircular canal, unlike the posterior and superior canals which lie deeply embedded, juts out upon the floor of the aditus as a small ivory-like hump and is exposed to the suppuration process.

2d. Its exposure occurs at the point of greatest constriction between the tympanic and antral cavities, and at a point of great pathological activity. The canal is not only subjected to the action of pus but to the deleterious combination of pus under pressure.

3d. The aditus is an unyielding bony ring in the circumference of which the external semicircular canal represents one of its weakest points and it succumbs accordingly.

4th. The vulnerable point in the external semicircular canal is not at the extreme apex of its arch—the point of greatest exposure —but at a point several millimeters below the summit of its ivory-like cap. This is due to structural differences at the points mentioned and to the fact that the base of this ivory-like hump is encroached upon or surrounded by a mass of cancellated bone which is easily disintegrated and in these cases always diseased.

I have seen no instance in which the extreme apex of the arch has been eroded: in every instance in which the canal has been involved the entire cap has disappeared.

In chronic suppurative cases we frequently have the opportunity to observe the effect of this undermining process as it has attained to different degrees of encroachment upon the lumen of the canal.

We find that the prominence of the canal presents at its base a thin, sharp edge so undermined that it would appear that by placing the finger-nail beneath it we could separate it as a little thin shell.

The manner of invasion in these cases is evident—the labyrinth is opened by a process of gradual erosion from without.

Of the second class, the remaining six cases are illustrative. In these the labyrinth is involved in a true suppurative process, and its various compartments are filled with granulations or pus, or both. These cases are in every way more formidable, more destructive and widespread, and more highly infective than the first and require bolder intervention. The vestibule is the site of greatest pathological activity, and in every instance in which I have found the vestibule involved, the canal system and the cochlea, either in whole or in part, have been involved also. This suggests the oval window as the most common avenue of entrance in the second class of cases, and this is in accordance with the pathological findings

in the capsule as shown in the previous cases. In the true suppurative cases the oval window was perforated in every instance with one exception. We may also have in an individual case the two pathological processes mentioned at work at the same time.

Inasmuch, therefore, as the exploration of the canal system, involving to greater or less extent its sacrifice, is necessary in this class of cases, the route which we adopt resolves itself into the selection of that which from a manipulative standpoint is the most convenient, which least endangers the important structures in surgical relation, and which allows us to fully expose the limits of the disease. The only route which fully meets these requirements is posterior to the facial nerve, and the point through which it is safest to enter the labyrinth for reasons to be mentioned later, is the solid angle of the semicircular canals.

We prefer to enter the vestibule posterior to the facial nerve instead of anterior to it, for the following reasons:

1st. When the vestibule is involved the semicircular canal system, in the great majority of cases, is involved also. This necessitates its exploration, which entails its sacrifice.

2nd. The sacrifice of the canal system, even though it is not extensively involved, creates only an ephemeral disturbance, and adds no danger to life, as the canal system is not in important communication with the intracranial cavity except by way of the vestibule.

3rd. We secure the maximum amount of working room, the advantage of which can be appreciated only when we have attempted the actual operation; and we are better enabled to work in the axis of the petrous pyramid, which is the direction of greatest safety.

4th. We get a field unsoiled by blood and gain a clear conception of the condition within the vestibule, of the ampullary areas of the canals, and of the inner vestibular wall.

5th. The route anterior to the facial nerve does not permit an exploration of the canal system which we know as a rule to be involved, nor of the ampullary areas, nor of the major part of the inner vestibular wall, but only of its lower anterior portion—consequently fistulae through the inner wall of the vestibule may be overlooked; I have had one such case in which had I adopted the anterior route, I would have committed this error and sacrificed the patient.

6th. It is based upon the erroneous idea that drainage is the chief desideratum and will suffice. Mere drainage in this class

of cases will not suffice, for not only is the interior of the labyrinth involved, but its capsule is, as a rule, profoundly diseased and such portions of it must be removed. In addition the complicated system of channels represented by the labyrinth cannot be satisfactorily drained from any one point, as from an opening in the outer vestibular wall.

That drainage through a given point will not suffice is shown by the fact that in many of these cases the outer capsule contained multiple fistulae—yet with multiple fistulae present, drainage was not accomplished. The mere existence of multiple fistulae, properly interpreted, is a plea for more efficient drainage than can be accomplished by this means. The attempt to drain the labyrinth through an opening in the outer vestibular wall, while it may in certain instances succeed, is comparable to the attempt to relieve cases of suppurative mastoiditis by merely opening the antrum. We should make the effort to place the present operative procedure upon a thoroughly scientific basis from the first.

7th. From a manipulative standpoint the anterior route is far more dangerous; it is the more cramped; the anterior canal wall interferes seriously with manipulation in the upper portion of the cavity; we are working in a blind pit and are forced to do considerable work from without inward, the direction of greatest danger; first, as the dome of the jugular bulb may rise high, undermining the outer vestibular wall; and second, as the inner wall of the vestibule bulges outward at this point, making the anterior portion of the vestibule comparatively narrow from within outward, an injury to the internal auditory meatus is a much more probable accident.

What are the steps of the technique?

As a description of the complete exenteration of the labyrinth will embrace the various steps, that alone will be mentioned.

1st. A Schwartze-Stacke operation, by which we secure the maximum amount of working room, and in which we exenterate the bony angle included between the groove of the sinus knee and the under surface of the middle fossa, which better enables us to work in the axis of the petrous pyramid. We lower the facial ridge to its absolute limit, remove the fringe of bone on the anterior aspect of the facial ridge back to the descending limb of the facial nerve, remove the outer wall of the hypotympanum, and lower the level of the canal floor, securing by these steps the maximum exposure of

the outer wall of the vestibule, and the dome of the jugular bulb should it rise high.

As exploration of the cochlea may be necessary, it is important that we see the exact position of the carotid artery by the exposure of its canal. In consequence we shave down the convexity of the anterior wall of the auditory canal, remove the lip of bone overhanging the mouth of the tube and evulse the tensor tympani.

This exposes the tube to curettement by which we relieve the field of blood, and it gives the maximum width to the apex of the cavity. Should the consistency of the bone permit, the arches of the semicircular canals should be delineated. This enables us to work with accuracy. (Fig. II.) The cavity should be cleansed; the tube packed with adrenalin gauze; the field rendered bloodless; the instruments and hands resterilized.

We next remove the prominence of the horizontal semicircular canal; it is a treacherous structure. The cutting edge of the chisel is placed at a point below the summit, but well above the level of the fallopian canal, for this prominence separates along definite planes of cleavage, and as the outer lip of this semicircular canal is intimate with the fallopian, a fissure in the former may extend out into the latter. Should the plane of cleavage be on a level with or below the fallopian canal, the facial nerve may either be exposed by having its roof removed, as shown in Fig. X, or else completely undermined (Fig. V), under which latter circumstances we may definitely expect paralysis for it will ensue. The stroke should be made in a direction corresponding to the plane of the canal. The remaining canals are next uncapped, and the condition of the interior is noted. It will be found that the interior of the external semicircular canal most frequently of all shows pathological change. In opening the superior canal a curved gouge should be used. It permits the stroke to be made in the direction of safety. The danger arises from the non-support of this canal by solid bony structure.

Owing to the contrast between the dark interior of the canals and the brilliantly illuminated white bone surrounding them, the labyrinthine fluid may appear dark and be mistaken for blood or granulations; we should not be deceived by this illusion.

We next enter the vestibule through the solid angle of the semicircular canals by creating at this point a conical pit with its apex directed inward and gradually lowered until it enters the vestibule.

As it is necessary to remove the inner lip of the horizontal semicircular canal, during this step the chisel should be held perpendicular to the plane of cleavage, and under no circumstances should it impinge upon the outer lip, which is intimately associated with the fallopian canal, together with which it is left as a bridge carrying the nerve which spans the vestibule. (Fig. IV.)

The opening in the vestibule is now enlarged until a full exposure is obtained of this portion of the cavity. Its inner wall should be searched for fistulae. In enlarging this cavity no pressure should be made upon the bridge. For this reason a curette is a dangerous instrument. A small sharp gouge is safer.

Owing to widespread necrosis, it is necessary in some instances to sacrifice the bridge of bone carrying the facial nerve. To accomplish this without injury to the facial, we select a curette with its cutting edge turned backward, utilize the superior rim of the bone cavity as a fulcrum and shave off from above downward in a direction parallel to the course of the nerve, the roof of the fallopian canal, thus leaving the nerve exposed in its gutter of bone, from which it may be separated and lifted without injury. (Fig. VI.) Any filaments given off from the nerve should be cut and not torn from this structure, as unnecessary traumatism is committed. We next remove such portions of the bridge as are necrotic, but no more, for the nerve here represents a curve, and should it lose the entire support of its bony gutter, it apparently elongates and consequently sags or kinks and becoming enmeshed in this vicious position by the granulations, has its functions interfered with later.

In this condition the nerve stretches as an exposed structure from its point of emergence low down on the facial ridge, across the cavity of the vestibule to its entry into the inner wall of the tympanum, corresponding to a point above and anterior to the original site of the oval window. (Fig. V.)

In knocking out the inner edge of the bony ridge, which corresponds to the upper inner wall of the tympanum, the stroke of the chisel should not be made from behind forward in a direction parallel to the course of the nerve, but from above downward, or, from before backward; i. e., in a direction perpendicular to the course of the nerve and to the fallopian canal.

The reason for this is that the bone at this point tends to separate when struck from behind forward, along a plane of cleavage which crosses the fallopian canal, causing its fracture; as the fallopian

canal at this point represents the greater portion of an exposed cylinder (Fig. I), its fracture results in the facial nerve being encompassed by a little annulus or cylinder of bone, which may be slid up and down upon the nerve, just as a ring upon a finger. This complication is—so far as the integrity of the nerve is concerned—a formidable one, and it requires the greatest patience to remove the little annulus without causing injury to the facial. To accomplish it we steady the annulus with mouse-tooth thumb forceps and with a pair of ronguers—the jaws of which are accurately apposed—crush it in a direction parallel to the course of the nerve.

To avoid this complication, it is only necessary before attempting the removal of the upper portion of the bony ridge that the fallopian canal should have been thoroughly converted into a gutter, by shaving off its roof, as mentioned, and by making the stroke in the direction indicated. Should the accident now occur, the nerve can be easily freed from the little mass of bone by slipping it through the open side of the annulus, or cylinder.

Should we have to expose the nerve as mentioned and shown in Fig. V, no attempt should be made to retract it or draw it to one side for the sake of gaining room for manipulation, as this is unnecessary; for the fallopian canal at the points at which the nerve emerges from and enters the bone is represented by sharp, serrated margins, against which if the nerve is drawn it may be lacerated at its fixed points. The sharp spicules should be removed so that the margin of the canal at these respective points is represented by an even rim.

The next step is to expose the anterio-inferior cavity of the vestibule by removing the posterior aspect of the promontory and the outer vestibular wall. (Fig. VII.) We select a gouge, the width of which corresponds to the distance between the round and oval windows. The cutting edge straddles the little bridge of bone separating these two openings, and the stroke—which is slight, for the posterior aspect of the promontory is brittle and fractures easily—is directed from above, downward and forward in the direction of the first cochleal turn. We must at this step think of the possible position of the dome of the jugular bulb.

The greatest care should be taken that the cutting edge of the chisel does not cross the cavity of the vestibule and impinge upon its inner wall, for the inner wall at this point bulges outward and is separated from the internal auditory meatus by a brittle partition of

bone not more than 1-32 of an inch in thickness. (Fig. XVI.) Should the inner wall be fractured, we lose cerebro-spinal fluid and aside from the inconvenience caused by this accident, the infected operative cavity is placed in direct communication with the intra-cranial cavity and the patient's life is jeopardized; meningitis will probably result.

As the cerebro-spinal fluid is under pressure and is of low specific gravity, gauze does not act as an efficient plug to the rent; sterile wax is preferable.

By enlarging this opening, we secure, with the previous steps, the full exposure of the vestibular cavity and the beginning of the first cochleal whorl. Should we now find that the disease has invaded the cochlea, and this is the rule in the second class of cases, we must continue the exploration of the cochlea until we have followed the diseased process to its legitimate end.

We next remove the roof of the first cochleal whorl from behind forward, exposing its interior to a point just short of the carotid eminence. (Fig. V.) The instrument of preference is a thin, sharp gouge with no shoulder, the width of which is slightly greater than that of the cochleal whorl. Four structures are to be avoided; the dome of the jugular bulb below, the eminence of the carotid canal in front, the base of the modiolus and the internal auditory meatus internally. The danger to the first two is slight; to the second two it is imminent, and injury to these structures is to be avoided only with the exercise of care.

By confining the removal of the cochleal shell entirely to the roof of its first whorl and not allowing the gouge to impinge upon the inner wall of the cochleal turn these last dangers may be averted. From the carotid artery in front, the cavity of the first cochleal turn is separated by a hard cuff of bone which serves the purpose of an efficient bumper: though thin this partition is sufficient. (Fig. XII.)

If we now find that the limit of the disease has not been reached, we must explore the remaining cochlea. This constitutes by far the most difficult and dangerous step of the procedure; for the cochlea, which represents an extremely small cavity encased in a brittle shell of bone, is hemmed in on all sides by structures which we cannot afford to injure. In front is the carotid artery, below the dome of the jugular bulb, behind the internal auditory meatus, above and behind in immediate proximity to the second half of the

first cochleal turn, the knee of the facial nerve. A circle a quarter of an inch in diameter could be so placed as to pass through the majority of these structures.

Were the above factors the only ones to be considered, it would be comparatively easy to select a point on the cochleal shell, which from dead house work we had found to be a safe one, and open its cavity. But the difficulty lies in the fact that within this shell of bone is contained a structure which from its position is exposed to injury, and which from a surgical standpoint is the most treacherous of the internal ear; I refer to the modiolus.

Before approaching the cochlea it will be well to consider certain anatomical features of the modiolus which are of surgical importance. The modiolus represents a small pyramid of bone seated upon the internal auditory meatus, decreasing in size from base to apex. Its apex is its weakest point, but the next weakest point is not immediately below its apex, but at the extreme base; for its base is excavated as shown in Figure XI (Politzer), by the internal auditory meatus, and the pyramid consequently rests upon a mere rim of thin, brittle bone. If the chisel is applied to the pyramid well above its base and a stroke made, the fracture does not take place at the point of applied violence but at its base; when this occurs the pyramid fractures completely round the circumference of its base and separates as a single piece of bone. (Fig. XIV.) The internal auditory meatus is consequently opened throughout its entire circumference, and as the diameter of the base of the pyramid, or the rim of bone upon which it rests, is about 1-6 of an inch, the loss of cerebro-spinal fluid is rapid. The failure to appreciate the surgical importance of this anatomical feature of the modiolus resulted in the death of the first patient.

If we now examine the modiolus with a strong convex lens we see that the pyramid has an outer casing of brittle bone and a core which is porous, made so by canals running from base toward apex for the passage of various structures. These canals are not completely filled by the structures which they contain, and this permits the cerebrospinal fluid to penetrate out into the modiolus.

During the second operation in which the modiolus had been removed well down toward its base it was noticed that a sepage of cerebrospinal fluid took place through its stump; the intra-cranial cavity had been placed in gross communication with the infected cavity in an altogether unexpected way through the afore-mentioned

channels, and the failure to appreciate the significance of this structural character of the modiolus resulted in the death of the second patient. In this case the infection could be traced along the cochlear branch of the auditory nerve. It now became important to determine how far down from the apex toward the base the pyramid could be removed without putting the intracranial and operative cavities in gross communication; for in dealing with the anterior half of the cochleal cavity it is absolutely necessary to get rid of a portion of the modiolus.

It can be shown upon increasing the tension of the intracranial fluid in a cadaver by injection, or in the living subject by pressure over the internal jugular vein, under which circumstances the cerebrospinal fluid will penetrate as far out as possible into the modiolus (i. e., grossly), that the pyramid may be removed from the apex toward its base, down to a point corresponding to the termination of the first cochleal whorl without causing the loss of cerebrospinal fluid; i. e., without placing the operative and intracranial cavities in gross communication. As will be shown later, this suffices for the complete exploration of the anterior half of cochleal cavity.

In approaching, therefore, the cochlea, we must remove its shell in such a way as not to injure the modiolus. We select a point in the cochleal shell corresponding somewhat to the apex of the cochleal cavity, and with a thin sharp gouge shave it down until the dark interior of a cochleal whorl shows through the thin lamella of bone. The stroke should be made from above downward and forward in a direction corresponding to that of a cochleal whorl.

Not infrequently the shell of the cochlea is scalloped and the position of the cochleal turns roughly indicated. In this way a window is created in the cochleal shell (Fig. X), and we now enlarge this window, completely exposing the upper portion of the cavity. (Fig. IX.)

In enlarging this window the small gouge is the instrument of preference; an attempt to insinuate a very fine curette beneath the opening in the cochleal shell causes its back to impinge or press upon the modiolus, which sticks up as a little tent pole in the cavity, and this may result in the fracture of the pyramid at its base. The gouge merely removes the shell without endangering the pyramid.

To expose the second half of the first cochleal whorl it is necessary to remove the apex of the pyramid down to a point correspond-

ing to the termination of the first cochleal whorl, as shown in Fig. XII. This done, we can look down over the stump of the pyramid, upon the roof of the second half of the first whorl, which is indicated by the probe in Fig. XII, and with a small gouge carefully break through its roof from above as shown in Fig. XIII, exposing in this way the entire interior of the cavity of the cochlea. In breaking through the roof over the last portion of the first whorl we are in direct relation to the facial nerve and the internal auditory meatus, both of which must be avoided.

The probe in Fig. XII is wound round the base of the modiolus merely for the sake of illustrating the relations of the roof of the second half of the first whorl. An attempt to withdraw it may cause the modiolus to fracture; such a step, of course, would not be taken during actual operation.

In one instance I have had to exenterate the major part of the petrous portion of the temporal bone and the following points have impressed me:

1st. That the petrous pyramid in this particular instance was pneumatic; we would expect to find such extensive involvement only in bones of pneumatic structure for reasons which are evident.

2d. Where removal of the petrous pyramid is necessary it should first be exenterated; later the shell removed; otherwise the dura of the posterior and middle fossae bulge into the wound, constrict the operative field which is extremely deep, and close it to successful view. Retraction of these structures is then necessary, but the best retractor is the shell of the petrous pyramid, as it commits no trauma, occupies no space, and is accurately applied to the surfaces to be supported.

A second reason for first exenterating the petrous is that the dura enclosing the superior petrosal sinus is firmly attached to the upper margin of the posterior aspect of the pyramid and separates with reluctance from this uneven lip. In consequence the vessel may be torn; sharp hemorrhage follows which is difficult to control, as it is not easy to place a compression plug between the dura and the bone, as the two do not readily separate. If the accident is to occur, it is more convenient that it should occur toward the end of the procedures.

We should attempt to save the facial nerve by preserving a bridge of bone extending from the lower portion of the facial ridge

FIG. 1.

FIG. II

FIG. III

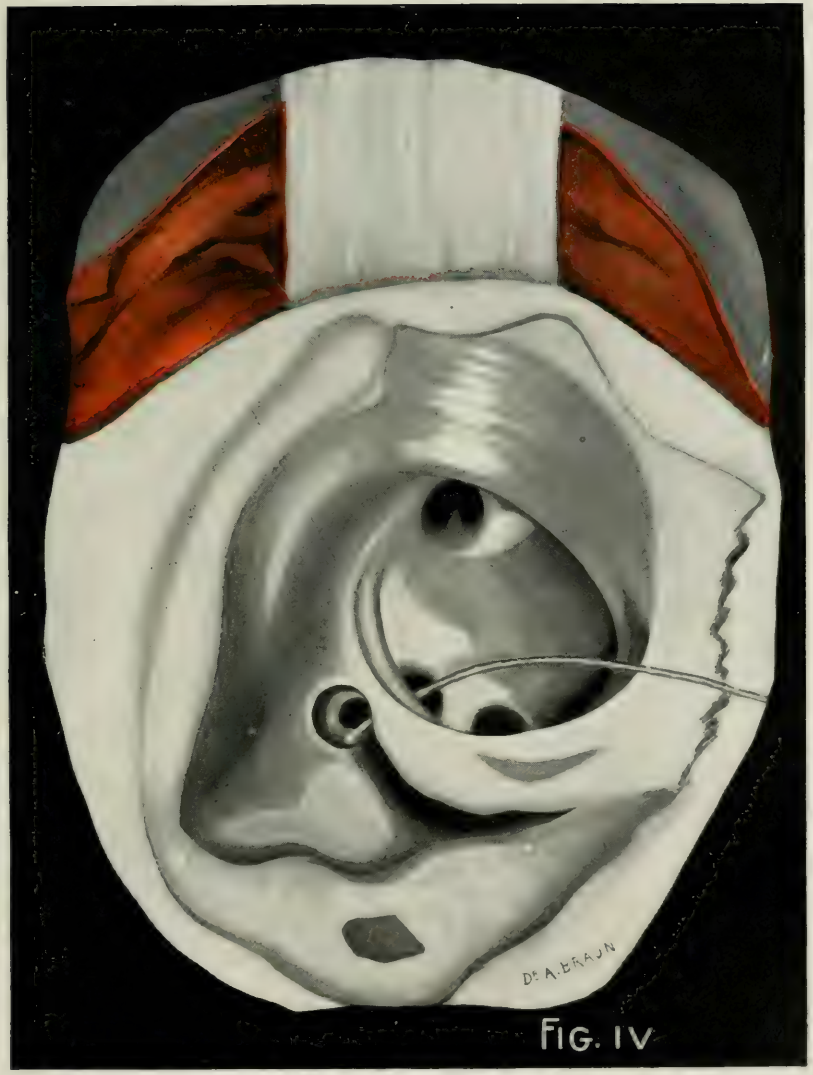


FIG. IV

FIG. V

FIG. VI.

FIG. VII

FIG. VIII

FIG. IX

FIG. X

FIG. XI.

FIG. XII

FIG. XIII

FIG. XIV

Fig. XV.

FIG. XVI.

to the internal auditory meatus; with patience this may be accomplished and, in the instance mentioned, was accomplished. Even though the nerve be sacrificed, we should still attempt to preserve the bridge, the purpose being to give the dura of the middle fossa a seat upon which it may rest that it does not sag deep into the wound and become sealed in this position to the granulation bed springing from the floor.

When the dura is not supported it sinks and shuts off the apex of the operative cavity, rendering this portion difficult to handle, and it further obstructs drainage. No amount of pressure by packing with gauze suffices to maintain it in a proper position, as the gauze soon becomes soaked and its supportive influence lost. Should, for any reason, reoperation be necessary—and we must remember that dyscrasiae may underlie such extensive destruction, and portions of necrotic bone may remain which circumstances at the time of operation either cause to overlook or to abandon—the dura sealed in its vicious position constitutes an efficient barrier to further operative interference. For this reason the operation should be thorough and all dead bone in the deep, apical portion of the pyramid removed. When the bridge is sacrificed the nerve, which represents a curve, is exposed over a considerable portion of its length and it kinks and becomes enmeshed later in the granulation bed; this interferes with function.

It is dangerous to attempt to remove sclerotic portions of the pyramid with the rongeur. The force requisite for a bite of the forceps is great, the bone suddenly gives and as the jaws of the rongeur spring together, sharp slivers of bone are thrown with violence against the tightly stretched dura of the cerebellum, which makes an excellent target to be perforated. I have seen the posterior fossa opened in this way.

It is highly important to be familiar not only with the surgical anatomy in the operative position, but to know the consistency of the bone; to know the result that will follow a stroke of the chisel when made in a definite direction; for the bone of the petrous pyramid is structurally treacherous, changing rapidly from pneumatic to absolutely sclerotic structure, which may crack when struck far beyond the point of applied violence and in a direction contrary to that expected. We can obtain a practical familiarity with this important part of the technique only by repeated work upon the cadaver, and an attempt at a description of this part of the technique

is useless. Generally speaking, we should, when attempting to remove sclerotic portions of the labyrinth, make the stroke in the long axis of the cavity which the sclerotic bone covers. While this is not invariably true, it is as near as the facts can be reduced to rule.

In operations upon the labyrinth a point of interest is to be noted with reference to the carotid artery, when through erosion of the carotid canal the artery is exposed to view, there is, as a rule, no visible pulsation in the vessel; I have seen the artery exposed in eight cases (some in chronic suppuration), and in only one instance was pulsation to be detected. If pressure is made upon the vessel with an applicator wound with cotton and the lumen of the vessel momentarily constricted, slight pulsation may be elicited. It is a wise provision that the vessel does not forcibly pulsate at this point and impinge upon the sharp serrated margin of the eroded canal; the walls of the artery in the canal are thin owing to a lack of the necessity for their full development.

The carotid canal serves a two-fold purpose: First, As the exposed artery in the canal shows no visible pulsation, or but a faint pulsation, it is evident that the carotid canal acts as a bumper to the pulse wave, diminishing its impact and shielding the brain from excessive shock.

Second, As the canal is rigid and does not permit the artery to dilate except within narrow limits, it has the practical effect of constricting that portion of the artery within its grasp. The effect is the same as when with the fingers we constrict the lumen of a rubber tube through which water is being forced by a rubber bulb, i. e. there is a tendency under these condition for the intermittent or remittent current to approximate a continuous stream. The carotid canal, therefore, not only protects the brain from excess of pulse impact, but it is a mechanism by which the inflow of blood to the brain is somewhat regulated and made more even and continuous. It is analogous to the hydraulic arrangement of the jugular bulb and sigmoid sinus on the venous side of the circulation, whereby an even outflow of blood from the brain is guaranteed.

During and following operation upon the labyrinth certain phenomena occur which are of exceeding interest.

If during anaesthesia we produce intermittent pressure upon the capitulum of the stapes, we may in certain instances induce nystagmus, which—should it occur—would rather point to the fact

that the labyrinth is functionating and not grossly involved in a destructive process. This experiment may prove of some value from a diagnostic standpoint in determining labyrinthine involvement, when the capsule shows no fistula.

If during operation we irritate with a probe the interior of the semicircular canals we may at time induce nystagmus. In the cases which I have seen the nystagmus has been bilateral and in the horizontal plane, regardless of which canal was irritated. The irritation of the inner vestibular wall gives a similar result.

The ampullary areas of the semicircular canals are more sensitive to irritation than their arches.

The upper posterior portion of the inner vestibular wall appears to be its most sensitive part.

A nystagmus which prior to operation is very marked may not disappear under ordinary surgical anaesthesia until the semicircular canals are removed and the vestibule opened, when it may entirely cease.

If the horizontal semicircular canal is by accident injured as during the acute mastoid operation, vertigo, vomiting, and nystagmus may appear and may be marked; again, these symptoms may be entirely wanting. As a rule, the disturbances which follow the injury are exceedingly slight and ephemeral, and consist of horizontal nsytagmus—most marked when the eyes are directed to the opposite side, and slight vertigo with a tendency on the part of the patient to fall away from the labyrinthine lesion. Profound disturbance of hearing, as a rule, follows.

If the operation has encroached to any extent upon a functionating labyrinth, particularly in cases where both the canal system and the vestibule are removed, disturbed equilibrium, nystagmus, and vomiting, which is accompanied by nausea, as a rule, follow. The vertigo is more pronounced when an attempt is made to assume the sitting posture, and closure of the eyes augments the disturbed equilibrium. The patient, as a rule, tends to fall to the side opposite the labyrinthine lesion.

At first the nystagmus may be of marked rotary character—the globes rotate beneath the closed lids, and nystagmus is independent of an attempt to fix the eyes upon an object. Nystagmus may be present in whatever direction the eyes assume. In a few days a marked decrease occurs, and as a rule, though not always, the great-

est excursion of the globes is in the horizontal plane. At first the oscillatory movements may be fine; later they are coarse, sluggish and jerky. The apparent fineness or coarseness of the movement appears to be dependent upon the rapidity of oscillation. The nystagmus, as a rule, presents the following features:

1st. It is most marked when the patient looks to either the extreme right or left lateral position.

2d. When the eyes are directed to the upper vertical meridian, slight nystagmus may be observed, which, at first may appear vertical in direction, but upon careful inspection a slight rotary movement will be seen. This latter is possibly due to the action of the extrinsic muscle of the eye.

3d. When the eyes are directed to the lower vertical meridian, the nystagmus is slightest, and, as a rule, absent, even though present in other meridians.

4th. The nystagmus is most marked when the eyes are directed to the extreme lateral position of the normal side. Even after the nystagmus has totally disappeared in all other meridians, it may still be made manifest when the eyes are turned from the labyrinthine lesion. Occasionally the patient tends to fall to the side of the labyrinthine lesion and not to the opposite side. When this occurs, the nystagmus may be most marked when the eyes are directed to the extreme lateral position of the operated side, or equally as marked as when turned to the normal side. There appears to be some harmony, as to direction, in which disturbed equilibrium and nystagmus are most manifest.

The vertigo and vomiting which follow operation upon the labyrinth, cease, as a rule, in a few days, and disturbed equilibrium and nystagmus soon subside. The patient, however, when attempting to walk a straight line, may show for some weeks or months subsequent to operation a tendency to deviate from the involved side; he may so tend to fall immediately after operation. In other cases all disturbances of equilibrium disappear in a few days. This tendency to fall or to deviate from the side of the labyrinthine lesion occurs in cases in which the horizontal canal alone is removed, in which the canal system and vestibule are both removed, or in which the entire labyrinth is removed. In some cases a slight nystagmus may be observed months after the operation; it may persist for years.

In some instances where the nystagmus is not of sufficient degree to be manifest when the eyes are directed to infinity, it may be made

so by having the patient suddenly converge and accommodate, though this is not the rule.

The primary direction of the oscillatory movements is away from the labyrinthine lesion, i. e., if the left labyrinth is involved the direction of oscillation is from left to right, or vice versa, no matter in what direction the eyes are turned; I have seen no exception to this.

The nystagmus and vertigo as above described are in no sense characteristic of the labyrinth; I have seen the same phenomena occur in cerebellar abscess, the location of which was in immediate proximity to its central lobe.

Upon the removal of the semicircular canal system, the vestibule, and the lower half turn of the first cochleal whorl, I have seen nystagmus, vomiting, vertigo, disturbed equilibrium, and intense high-pitched hissing noise, which were distressing before operation, immediately cease.

As to why the destruction of the semicircular canal system and vestibule may in the one instance be productive of vertigo, vomiting, nystagmus and disturbed equilibrium, and in the other cause these same symptoms to quickly disappear, I do not know, but even where these symptoms follow operation upon the labyrinth and are marked, they, as a rule, so quickly subside that it is evident first, that the semicircular canal system is not an indispensable factor to the preservation of equilibrium; and second, that we can scarcely attach to it the importance of being the central organ of the statical sense, as its loss can be so quickly compensated for and in some instances seem to produce no disturbance whatever; it evidently is a part of more or less importance in the great circuit governing equilibrium, but it is by no means an indispensable part, nor is the entire labyrinth indispensable.

Following the removal of the major portion of the labyrinth, conjugate deviation of the eyes may occur, the direction assumed being upward and outward toward the operated side, as if the eyes were attempting to see the labyrinthine lesion. It should be mentioned, however, that in the two cases in which the phenomenon was observed, cerebro-spinal fluid was lost in both. This suggests that the conjugate deviation may have arisen from other causes than the destruction of the labyrinth. The conjugate deviation was noticed immediately upon the patient's emergence from anaesthesia.

Following the removal of the deep portion of the petrous pyramid, we may notice upon the patient's emergence from anaesthesia

that both pupils are dilated ad maximum. This dilation probably results from disturbance of the sympathetic, at the deep, apical portion of the pyramid.

The pupils, though dilated ad maximum, may react to both accommodation and to light. The dilatation gradually disappears after several weeks.

Following the exenteration of the petrous portion of the temporal bone, probing or pressure in the immediate vicinity of the stump of the auditory nerve may produce vertigo, nystagmus, and the subjective phenomenon of intense noise, which noise is referred to in the involved ear. The trunk of the auditory nerve is capable of interpreting mechanical stimuli, as sound, and from the case which I had the opportunity to observe, I am inclined to believe that the trunk of the auditory nerve is also sensitive to waves of sound.

Following the exenteration of the labyrinth, the patient may be exceedingly sensitive to noise and to loud musical sounds. The sense of harmony may be lost and after several months regained. The patient, if subjected shortly after operation to loud musical sounds, as the playing of the piano, may become intensely dizzy, the vertigo attaining to such a degree that it necessitates his sitting that he does not fall.

It would appear that we receive through the auditory apparatus certain physical impressions which, when transmitted to higher centers, act as supporting influences to the preservation of station; that these, when not balanced by similar impressions acting upon the other ear cause, so to speak, an auditory imbalance; that the disturbance is due to the lateralization chiefly to one side of those impressions, which give rise to the subjective phenomenon of sound is questionable, inasmuch as the patient who was totally deaf before operation experienced then no such disturbance under similar conditions.

The phenomenon is suggestive of the great variety of factors which may act through the medium of the auditory apparatus and quickly disintegrate and completely disappear. The nerve should contribute to the preservation of that condition which we speak of as the state of equilibrium—it gives us an insight into the intricacy of the function of the auditory apparatus.

Following the removal of the entire labyrinth, tinnitus may still persist in the involved ear; its character, however, may completely change, and in all the cases the tendency has been—even where the

tinnitus before operation was intense and of high pitch—for it to assume a low, buzzing, inoffensive character after operation. Later the tinnitus may entirely cease.

When the facial nerve is exposed throughout its entire circumference, we may with confidence look for paralysis upon the patient's emergence from anaesthesia. The paralysis deepens and in forty-eight hours becomes complete. It persists from four to five months, even though no injury is done the nerve other than its exposure to the wound and its fluids, and though the reactions of degeneration may appear, the face under massage, and with time, regains its nearly normal expression; I have seen, however, no case in which the function has been so completely restored that the variation in the two sides could not be detected upon the act of smiling. When the nerve is exposed in the manner mentioned, it is difficult at the time of operation to make it respond to ordinary mechanical stimulation, and moderate heat does not affect it.

The facial nerve is structurally very delicate. I have seen a nerve after several days' exposure to an infected cavity which had been irrigated but twice with a 1-10,000 solution of bichlorid of mercury, be protected by sterile vaseline and subjected to no pressure by the packing and the dressing should be changed sufficiently often to keep the cavity clear of pus.

Subsequent to operation, meningitis may occur. It manifests itself in from forty-eight to seventy-two hours. It is characterized by the usual features, with the exception that the sensorium is remarkably clear till near the end; the termination is rapid. Though the postmortem may show a diffuse leptomeningitis, the cerebellar fossa is the site of greatest pathological activity. The route of invasion may be apparent, and in one of my own cases was along the course of the auditory nerve.

When the internal auditory meatus is opened, the rapidity of escape of cerebro-spinal fluid suggests the proximity to the canal of large quantities of cerebro-spinal fluid, as if the cerebellar fossa contained or was in direct communication with a relatively great amount. In the period of approximately a minute, the cavity represented by the Stacke-Schwartze excavation may fill as many as three times.

Following the too rapid drainage of the cerebrospinal fluid from the cerebellar fossa the following symptoms may appear: a small, rapid, irregular pulse; embarrassed respiration, which may assume

a Cheyne-Stokes character; an anxious, concerned expression, with cold extremities and pinched, livid features; marked restlessness.

These symptoms may be of such alarming character that death seems imminent, but with a lessening of the escape of cerebro-spinal fluid the whole picture may in the course of a few hours so completely change that the patient appears quite normal. These symptoms are undoubtedly due to a too rapid drainage of the water-bed of the brain, whereby the important nerve centres at the base, having lost their elastic support, sink and are brought into undue contact with the floor of the skull.

The quick disappearance of these symptoms upon the cessation of the escape of cerebro-spinal fluid forbids any other than a mechanical interpretation.

Following operations upon the labyrinth, large quantities of sugar and acetone may appear in the urine, and the clinical picture of diabetes may rapidly develop. These phenomena suggest that operation upon the labyrinth may be productive of a profound systemic shock which does not manifest itself in the usual disturbances of temperature and pulse. The causative relation, however, between shock and diabetes is highly suggestive of the major character of the labyrinthine operation.

In three cases where the major portion of the entire labyrinth was removed, including the semicircular canal system, there appeared to be after a lapse of over two years in the first, one year in the second, and nine months in the third, no sign whatever of muscular atrophy of either side of the body.

Where the destructive process in the labyrinth has existed for a long time and the irritative stage is passed, the organ, so to speak, is functionally silenced and shows no sign of life; its exenteration is followed by no disturbance whatever of equilibrium; no vomiting, vertigo, or nystagmus occur, as the compensating factors, whatever these may be, have made good the deficiency.

That the disturbance of equilibrium, nystagmus, etc., which follow the removal of portions of the labyrinth are due, to some extent, to the loss of these parts is highly probable, inasmuch as these symptoms continue, even though the interior of the labyrinth is thoroughly cocainized. That these symptoms, however, are not altogether due to the loss of these parts is evident, inasmuch as under anaesthesia, mechanical irritation of the interior of the vestibule may produce nystagmus, as may also pressure upon the ampullary

areas of the semicircular canals. Pressure upon the stapes may give a similar result. Further, if we irritate with a probe certain portions of the labyrinth subsequent to operation, we may in some instances cause the patient to fall, by producing an intense vertigo. Nystagmus may also be induced or augmented by pressure over certain areas. It is highly probable, therefore, that the disturbances of equilibrium which follow the removal of portions of the labyrinth are due not only to the loss of the parts destroyed but also to peripheral irritation, to which the interior of the labyrinth is subjected. It is only upon this double basis that we can explain certain phenomena in connection with these cases.

For instance, in Case X of the writer's series, disturbed equilibrium, vertigo, vomiting and nystagmus which were intense and distressing before the operation, immediately ceased upon the removal of the semicircular canal system, the vestibule and the lower half of the first cochleal whorl. If, therefore, these disturbances are due to the non-activity of the canal system, and not to irritation, why should the patient when in possession of these parts have exhibited intense disturbances of equilibrium, and after their removal, which insured their non-activity, have shown no disturbances of equilibrium whatever?

Following operation upon the labyrinth, the pulse may be both irregular and rapid and this disturbance may be independent of the loss of cerebrospinal or labyrinthine fluid; the condition soon rights itself.

From the following facts certain conclusions may be drawn, which are of some practical interest in that they show the difficulties attending labyrinthine localization. If we remove the arch of the horizontal semicircular canal, there may follow, vertigo, vomiting, nystagmus and disturbed hearing.

The removal of the entire canal system and the vestibule may be followed by the same phenomena.

The accidental removal of the stapes may produce vertigo, vomiting, nystagmus, disturbed equilibrium and disturbed hearing. The destruction of portions of the vestibule and cochlea without destruction of the canal system produces the same phenomena. It is now evident that if we deny to these various phenomena, representing several different functions disturbed, the entire labyrinth as the anatomical seat of each (for these phenomena follow no matter

what portion of the labyrinth is destroyed) we are led to the following conclusions, which seem to be supported by clinical evidence:

1st. That if the integrity of the labyrinth is encroached upon at any point, the function of hearing, as well as the non-acoustic functions, is seriously interfered with.

(If the encroachment has been gradual and there is no apparent disturbance of equilibrium, etc., it is due to compensation.)

2d. That the disturbances which follow the destruction of a given portion of the labyrinth are not necessarily to be attributed to the loss of the parts destroyed.

3d. That the destruction of the labyrinth at a given point affects the function of distant parts as well. If, for instance, we destroy the entire labyrinth with the exception of the cochlea, or, if we merely remove the arch of the horizontal semicircular canal, we are at a loss to know whether to attribute many of the disturbances which follow to the loss of the parts destroyed or to the irritation of the parts remaining.

It therefore follows that an attempt at labyrinthine localization based upon symptoms will always be subject to the grossest error, and that the attempt to determine the functions of the various departments of the labyrinth by the experimental destruction of these will be subject to the same error.

The difficulty of experimentally determining with accuracy the functions of the various parts of the labyrinth with a view to labyrinthine localization, and this is here alluded to merely on account of its surgical interest, is due in large measure to the anatomical arrangement of the labyrinth, the essential feature of which is, that it is suspended in a fluid medium which is *continuous*. A disturbance of the medium at any point is transmitted throughout the entire apparatus and results in the disturbance of distant parts.

While this particular anatomical arrangement complicates the study of the physiology of the labyrinth, it is an essential attribute of an organ which embraces several distinct and separate functions, which are correlated to the extent that each is influenced through the same common medium and to some extent by the same primary cause —waves of sound.

The labyrinth is an example which illustrates the conservation of energy in the evolution of an organ in that the anatomical bases of its

several functions, which in reality represent different organs, should have been so grouped together and suspended in a common medium as to make common use of that medium. This arrangement is evidence of the influence which waves of sound exert upon the non-acoustic as well as upon the acoustic labyrinth and a practical example of this is illustrated by the case previously mentioned where the patient when subjected to loud musical sounds, after the exenteration of the entire labyrinth, was made so dizzy as to necessitate his sitting, his equilibrium having been profoundly disturbed.

While it is the purpose of the present paper to treat of only those conditions which have a surgical bearing, there are many facts in connection with the cases which are of interest in that they are in contradiction to certain theories which have gained a more or less widespread acceptance.

DISCUSSION.

Dr. Frederick L. Jack, of Boston, cited two cases. In the first purulent involvement of the labyrinth followed within a week an acute middle ear suppuration. All the symptoms disappeared in a short time with the exception of the profound deafness, which is always likely to remain. In the other case the entire osseous labyrinth, a most perfect specimen, was removed as a sequestrum. There was no history of vertigo or vomiting. In chronic cases this exfoliation might occur without any direct symptoms such as would be found in an acute invasion.

Dr. F. C. Ward, of Plainfield, N. J., cited a case of serous meningitis following otitis media. Dr. Richards suggested the possibility of draining the subarachnoid space through the internal ear. This was done and the patient lived seven or eight days subsequent to the operation with almost complete relief from pain.

Dr. Wells P. Eagleton, of Newark, N. J., said he had observed nine cases of labyrinthine suppuration, seven of which had been operated upon. His chief interest in the subject had been with reference to the matter of diagnosis previous to operation. The inability of patients to jump with closed eyes was an important symptom, which would enable one to make a diagnosis of labyrinthine disease without other symptoms. He detailed briefly the physiology of the apparatus of equilibrium. If from any cause there was disturbance of the semicircular canals there would be, first of all, symptoms of vertigo and nystagmus. This was generally associated with vomiting, and patients think the disturbance of equilibrium due to the gastric condition.

Dr. Ewing W. Day, of Pittsburg, Pa., called attention to one point in technique which he had found useful, viz., rendering the field of operation bloodless. He had used a subperiosteal injection of normal salt solution with adrenalin, 1-1000, with the result that it gave an almost clear field, free from oozing, and leaving everything so that it could be seen.

Dr. Edward B. Dench, of New York City, called attention to the fact that labyrinthine infection might come through the cranial cavity itself. While almost all cases followed chronic inflammation, the involvement of the labyrinth might occur as the result of an acute process. Sudden disturbances of hearing, together with nausea and vomiting, should make one suspicious of labyrinthine disease. The mere fact that there was vertigo and nystagmus need not direct the attention to the labyrinth. The only point to be observed with reference to the nsytagmus was its character. He did not advocate the radical operation unless there were symptoms to warrant it, such, for example, as pus coming from the oval window, the presence of sequestra of bone, or symptoms of meningitis in addition to the ear symptoms. If there were simply a small opening in the horizontal semicircular canal he would delay extensive operation; he would be content to clear out, pack, and wait. Granulations around the stapes should always be removed. Not infrequently the stapes would come away with the granulations. The oval window should be very carefully explored with a small, delicate probe, because nature may already have walled off the labyrinth. If pus exudes, the labyrinth should be opened. There was nothing to be said about the technic of the operation after the splendid presentation of the subject by Dr. Richards.

Dr. Reik, in closing the discussion, maintained that destruction of the cochlea means destruction of the hearing power, and would so maintain until something more than the subjective sense of the patient was produced to the contrary. Stimulation of the stump of the optic nerve would not produce sight, and so stimulation of the stump of the auditory nerve would not produce hearing.

PAPER:

PATHOLOGY OF THE FAUCIAL TONSIL.

JAMES A. BABBITT, M.D.

The assignment of a topic upon the pathological conditions of the tonsil presents a field most comprehensive in its scope, but its fortunate limitation to a surgical symposium in certain degree diminishes this range. While the tonsil is comparatively simple in histological structure, its important position at the alimentary inlet, close relation to cervical lymphatics, remarkably free vascularity, and exposure to constant impact from foreign material, render it anatomically and functionally of considerable significance.

These minor groups of adenoid tissue comprising the so-called adenoid ring, omnipresent and familiar almost to a degree of contempt, save in relation to the anatomical interference and retardation of functional development, still in large measure defy scientific investigation as to their exact import in the body economy, and still leave indiscriminate surgical interference a question of justifiable debate.

Most admirable and painstaking work has been done upon this investigation by Packard, Wood, Swain, Wright, Browne, Hall, Tilly, Kyle, Allen and others, yet notwithstanding their splendid contributions to this field of literature, the tonsillar function still remains a matter of opinion.

In view of noteworthy studies already published on cryptal degeneration, lympho-genetic function, tubercular significance, focal areas for bacteria, vaso-motor connection, etc., it would seem best in this paper, after brief outline description of anatomical and physiological characteristics, broadly to classify and describe pathological conditions present, and finally, perhaps, discuss in more detail some special topic of practical application in routine surgery of this part. Inasmuch as chronic hypertrophy and its resultant effects are most significant upon general metabolic activity, the pro-

gressive change of the tonsil during the developmental period would perhaps be primary in importance, and next in order the systemic effects of cryptal absorption.

SUGGESTED ORDER OF STUDY.

No. 1. Brief review of anatomical and physiological characteristics.

No. 2. Classified description of pathological conditions acute and chronic.

No. 3. General effect of hypertrophic enlargement in the naso-pharyngeal area.

No. 4. Correlated physiological influence upon general metabolism.

No. 5. A comparative study of hypertrophic changes at stated age intervals.

NO. I.

Anatomically, the faucial tonsils, lying just internal to the angle of the jaw, between the palato-glassus and palato-pharyngeus muscles, consists of a mass of glandular tissue, flattened ovoid in shape, of the type of compound lymphatic glands, and in early life about three-quarters of an inch in general dimension. Each tonsil contains from twelve to fifteen cryptal orifices, formed by invagination of the epithelial layers of mucous membrane, and from these crypts numerous irregular communicating channels branch out into the subjacent parenchyma. In the walls surrounding the crypts are found numerous nodules, without, however, directly communicating ducts, the contents being discharged directly through the epithelial walls of the crypt. The pharyngeal surface of the tonsil is indurated from constant foreign impact, and crypt mouths become partially occluded by cicatricial bands of fibrous tissue in later life. The mass is closely adherent to the superior pharyngeal constrictor, often adherent to anterior and posterior pillars. Harrison Allen has called attention to the almost uniform presence of a sulcus dividing lower smooth from the upper cryptose position and a posterior velar portion. According to Piersol, the epithelium lining of the crypts becomes completely infiltrated with lymphatic cells.

Beneath the indurated cortex, a much softer parenchymatous portion exists, and closer study of lypmhoid follicles shows close anal-

ogy to Peyer's Patches and solitary glands of the intestine. Within the crypts the epithelium shows marked degenerative changes, and in adult life small cheesy pellets are often present, formed from accumulated secretion, and from time to time are ejected.

Attention should be called to the supra-tonsillar fossa upon and back of the tonsil covered by the plica-triangularis, inasmuch as this is a most important pathological focus.

The vascular supply is derived from the tonsillar, lingual, facial, internal maxillary, and pharyngeal arteries—and the nerve control by branches from the trifacial and glossopharyngeal.

Of important significance is the lymphatic outlet into upper deep cervical nodes, lying along the internal jugular vein, and thus communicating with the axillary and superior mediastinal spaces. Its embryological development has been most diligently studied by many authors, but is without the province of this paper.

In anatomical summary, then, tonsillar points of interest are:

No. 1. Mucous membrane and crypts.
No. 2. Interfollicular tissue.
No. 3. Lymphoid follicles and nodules.
No. 4. Fibrous tissue stroma.
No. 5. Vascular supply.

Physiologically the tonsil presents a far more difficult problem as the data available on functional importance are meagre, and many authors merely consider these glands adjucants to salivary work in deglutition. Wood, who has carefully covered the field, emphasizes the lymphogenetic possibilities; others the phagocytic-absorptive, etc.

Inasmuch as the tonsils contain closed lymphoid follicles, a first inference would be that they should be included in this category of the so-called ductless glands and be estimated with the thyroid, suprarenal and others. Indeed its embryological relation with the second branchial cleft would strengthen this assumption. Yet a study of the secretion fails to reveal anything commensurate with the renal, colloidal and other associated fluids. Its contribution to the saliva and lymphatic manufacture, partly chemical and partly physical in importance, probably limits its field in this direction.

The fact that toxaemic and other materials are frequently absorbed through the crypts, would bring next consideration to its possible share in alimentary absorption and nutrition. Its connection with the lymphatic channels is certain, and this possibility is

not remote, yet its surface is so comparatively small and extirpation shows so little disturbance that it seems almost inconsequential.

It has been suggested that the tonsils through moderate vasomotor reaction may serve some part in protecting the pharyngeal cavity, by influencing secretion. This, again, has no corroboration.

Inasmuch as the throat first receives pathogenic matter—admitted to the system per oval cavity—its protective and possibly antitoxic properties should be considered. Epithelial tissues in general have a positive selective resistance to toxic absorption, and the constant formation of leucocytes would indicate phagocytic action. The alimentary mucus membrane, universally glandular in type, preserves this resistance in uniform degree, as per evidence of the immunity to ptomaine contact in the intestines, and it is entirely possible that the free display of adenoid tissue in and about the pharyngeal cavity, and particularly in the tonsil with its specialized tissue formation, may contribute to this capability of resistance.

Newcomb has called attention to the constant ramification in the epithelial surface of the crypts as the tonsil atrophies, allowing various organisms to pass through the thin walls, again strengthening the theory of protective function during the period when all adenoid tissue is most marked.

In experiments upon colors rubbed into the hypertrophic tonsils, Goodale found them taken into the interfollicular space but not in the follicles. And attention has been called to the free phagocytic migration from the centre to tonsil surface functionally protective in destroying septic organisms so prevalent in the mouth.

NO. II.

Classified Descriptions of Pathological Conditions, Acute and Chronic.

The variance in terminology renders a definite classification of tonsillar affections somewhat difficult. By many authors the term lacunar has been substituted for the appelation "follicular," customarily applied to such a common condition of the tonsils, as distinguishing the cryptal inflammation from that involving lymphoid follicles.

A simple classification based as much on symptomatology as microscopic change might be as follows:

(A) Acute Types.

Inflammatory, superficial or catarrhal tonsillitis.

Lacunar or cryptic tonsillitis.
Parenchymatous tonsillitis.
Peri-tonsillar abscess.
Acute ulcerative tonsillitis (rare).
Membranous tonsillitis.
Herpetic tonsillitis.

(B) Chronic.

Chronic hypertrophy of the tonsils.
(a)—Fibroid.
(b)—Soft Adenoid enlargement.
Chronic lacunar tonsillitis.
Mycotic inflammation of the tonsils.
Tonsillar calculi and foreign bodies.
Traumatic and escharotic affections.
Rheumatoid tonsillitis.

No. 2. Neoplasms.

(1) *Benign tumors* of the tonsils.
Papillomata.
Fibromata.
Angiomata.

(2) *Malignant Tumors.*
Carcinomata.
Sarcomata.

In addition, cases of Lipomata and Lympho-sarcomata have been reported.

In all these various degrees of acute and chronic inflammation of the tonsils, bacterial germs most frequently involved are the pyogenic cocci, but these will be considered in the discussion of inflammations.

(a) In the acute catarrhal or superficial tonsillitis, as termed by Kyle, the inflammatory condition is that of a simple glandular mucous surface. First, Distention of blood vessels; second, exudation of fluid and diapedesis of cellular element from central current; and third, various degenerative changes in proximal fixed tissues and proliferation of tissue cells.

This simple inflammatory condition, with customary symptomatic changes of increased temperature, redness, thickening and modified nutrition, may undergo resolution or advance into more defined lacunar types, producing surface necrosis or involving deeper reticulated and lymphoidal structures of the tonsil.

In these inflammatory stages we note the marked phagocytic effort of the leucocytes to resist pathogenic micro-organisms, by massing themselves about, absorbing and digesting toxic elements.

(b) Acute lacunar (often termed follicular tonsillitis) like the catarrhal form in advanced stage, is characterized by considerable accumulation of inflammatory exudate in the crypt passages. This includes epithelial cells, leucocytes, bacteria, and various forms of debris, and in the chronic stages, masses of keratoid material formed by epithelial degeneration. Associated with this will be found marked degree of cell proliferation in adjacent tissues, and inflammatory exudate into parenchyma, producing infiltration of leucocytes and an oedematous condition of adjacent glands.

Paterson and Sawtelle demonstrated that the Supra-tonsillar fossa, which has almost direct connection with superior crypt and follicles, was the seat of primary entrance for tonsillar infection.

Microorganism presence in the tonsils have been investigated by many authors.

Streptococci—both forms, Staphylococci (aureus and albus) and the Diplococcus predominate; in lesser frequency the Pneumococcus, Kloebs Loeffler bacillus, Staphylococcus citreus, Micrococcus tetragonus, Micrococcus albus liquifaciens, Leptothrix fungus, and others, more sporadically. The essential qualifications for most bacterial growth are here present in the deeply lying crypts—warmth, moisture and nutritive pabulum—augmented by inflammatory change.

It has been shown by recent research that the tonsil is often the seat of primary and secondary tubercular deposit, while in general rheumatic fever and the exanthematous diseases the tonsillar condition is primary in many cases.

Allusion might here be made also to specific infection of syphilis and its attendant chain of ulcerative and necrotic conditions.

No. 3. Parenchymatous tonsillitis.

Beyond the cryptal structure of the tonsil we find secondary isolated nodes with an inner follicular parenchyma, and this divided by fibrous trabeculae passing from the capsule and carrying vessels. Associated with lacunar inflammatory change, this entire parenchymatous mass may become involved with characteristic exudate. This may readily resolve in time to tonsillar abscess with deep encysted pockets.

No. 4. Peri-tonsillar abscess is closely allied to the general parenchymatous inflammation, in fact, abscess of true tonsillar tissue most often commences in the peritonsillar area.

According to Shurly the first step in peritonsillitis is determination of the blood to the tissues caused by toxic action on blood vessels. In fact, anything seriously disturbing relations between internal and external capillary circulation may be an exciting cause.

Peritonsillar abscess, or quinsy, is so familiar to specialists and general practitioners as to require little description. Its process is essentially inflammatory involving surrounding pharyngeal and lymphatic tissues. A rapid infiltration and cell proliferation, marked effusion from vessels and lymphatic channels; leucocytes subject to chemo-toxic attraction, necrosis takes place and abscess points in line of least resistance, spreading freely if in outer thin tissues, but often requiring deep incision through body of tonsil when confined to tonsillar structure proper. The tonsillar cyst has also been described in this connection.

No. 5. Acute ulcerative tonsillitis, *per se,* is infrequent and is closely allied to necrotic areas secondary to aphthous deposit and the small ulcerations left upon removal of herpetic blebs.

No. 6. The membranous or aphthous is very similar to membrane produced by Kloebs Loeffler bacillus. The membranous deposit is continuous with that in the crypts, and may spread over the entire tonsil and neighboring pillars. It is in form a coagulation necrosis, producing surface ulceration of varying depth.

Superficial layers of mucous membrane are infiltrated with pus cells, exudate and bacteria.

It consists of grayish membrane, often bleeding if detached, and often an exanthematous complication. As has been noted, non-pathogenic bacteria become pathogenic under such conditions of weakened tonsillar resistance.

No. 7. Herpetic tonsillitis was early described, by Moure of Bordeaux, as consisting of small vesicles which break in twelve to twenty-four hours, leaving ulcers covered with whitish exudation.

It is likewise a coagulation necrosis of surface epithelium with vascular exudate.

The tonsillitis due to rheumatic diathesis is so closely allied to the catarrhal and parenchymatous types that we have not here given it special classification.

(B) Chronic Types.

No. 1. The omnipresent chronic hypertrophy of the tonsils may be either the true hyperplasia of all tonsillar structures or a hypertrophy of the connective tissue elements. It is an almost invariable sequence to repeated attacks of acute tonsillitis and probably has been most often investigated of all pathological conditions.

The appearance of the hypertrophied tonsil is characteristic. A large lobulated mass projecting well beyond the margins of the pillars with crypts freely visible, contracted or open in accordance with age and progression of hypertrophy.

Lennox Browne classifies these forms of hypertrophy as: 1. Preinsulated or Projective.

2. Cowled.

3. Lacunar, honey-combed or ragged and in morbid anatomy as—No. 1. Interstitial or sclerotic. No. 2. Lymphoid.

Bosworth thinks one-third to one-half of this number of cases arise from infectious diseases and places etiological order. First, scarlet fever; second, Diphtheria; third, Measles; fourth, Small Pox. Unilateral hypertrophy has frequently influenced suspicion of syphilitic origin or development of neoplasm.

Unquestionably the diathetic or strumous diathesis and the exanthematous diseases are in large part causative of this condition. Lachonnek claims that infection most often begins in Waldeyer ring, especially the faucial tonsil, and Packard reports five cases of endocarditis all preceded by attacks of amygdalitis.

According to Price-Brown Tonsillar Hypertrophies are due to:

1. Congenital Causes.
2. Diathetic Conditions.
3. Exanthematous Diseases.
4. Congenital Syphilis, Rheumatism, etc.

Its actual pathological change consists as stated either in general hyperplasia of all glandular elements with cell proliferation and infiltration, or marked overgrowth of fibrous tissue, trabeculae and subsequent contraction. Its associated pathologic influence will be discussed later.

2. Chronic Lacunar Tonsillitis.

This follows repeated attacks of the acute lacunar type, and inasmuch as the crypts are often partially obliterated by fine fibrous tissue trabeculae, caseous plugs of inspissated secretion (often keratoid mass) commingled with decayed food products and pos-

sibly bacteria are found in crypt pockets. Often these are extruded as firm almost caseous masses . (Wood claims a non-pathogenic state here.)

3. Mycotic inflammation is due to fungous mass, essentially a form of necrosis, and usually arising from the crypt walls, though often spreading to the surrounding surface and even pillars. The leptothrix bacillus is usually causal and the condition is comparatively benign.

4 and 5. Calculi, foreign bodies, traumatic abrasion, escharotic injury, etc., produce pathological changes of acute and chronic inflammation respectively and in this category might possibly be included drugs influencing inflammatory change such as Mercury, Antimony, Potassium Iodide, Arsenic, Copper, Lead, Zinc and Belladonna.

In reference to Calculi it has been maintained that the supra-tonsillar fossa is an especially vulnerable position for such concretions, that these vary from small specks even to an ounce weight, and resemble tartar on teeth.

Gruening states that all tonsillar concretions contain leptothrix and that this influences precipitation of lime in form of chalk, while others hold that tonsillar calculi consist principally of phosphate and carbonate of lime.

Neoplasms have been especially well studied by Browne, Hall and Tilley and the older continental investigators. Sufficient here to allude to main characteristics which resemble similar tumors in other glandular regions. Of the benign tumors the papillomata are wart-like excrescences emerging from the crypt walls or found at supra-tonsillar portal. The fibromata are usually pedunculated or sessile, polypoid in appearance and likewise protrude from crypt margin or adjacent tonsillar surface. In the malignant group carcinoma are of the epithelioma type, and may be squamous, columnar or alveolar, while sarcomata may be of simple type—such as round or spindle celled—and compound, as the angio-sarcoma, lipoma and myxo-sarcoma (lympho-sarcomata have also been reported).

The tonsils as portals of infection are elsewhere discussed in detail.

NO. III..

General Pathological Effect of Hypertrophic Enlargement in the Nasopharyngeal Area.

The general pathologic effect of chronic hypertrophy is almost directly that of respiratory interference.

Primary objective symptoms are localized functional obstruction, a muffled, nasal or cracked voice, difficult articulation, embarrassed deglutition, frequently a chronic hacking cough, high narrow palate-arch, constricted nares, drooping lower maxilla with faulty dental alignment, vitiated sense of taste, smell and hearing, dull and listless facies, stupid expression and thickened lips, mouth breaking and oral membrane impaired by exposure.

Of deeper and secondary significance are chest deformities such as pigeon or chicken breast, disturbed night rest with heavy snoring respiration, habit choreas and stuttering, foetid breath, increased susceptibility to bacterial invasion, increased tendency to contract bronchial affections, and general systemic mal-nutrition.

These and many other primarily functional and secondarily organic deficiencies, really pathologic in import, can be traced directly to the hypertrophied faucial tonsil.

NO. IV.

Next should be considered correllated physiological influence of this hypertrophy upon general metabolism.

Much of the foregoing enumeration rightly belongs to the category of this heading, but in addition emphasis should be placed on three points.

1. Direct relation of the tonsils to deep cervical nodes and general lymphatic system.

2. Remarkable vascularity in tonsillar mucous cortex and subjacent parenchyma.

3. Close relation to cervical sympathetics and consequent systemic influence of chronic irritation.

By reason of free vascularity no nasopharyngeal area more readily responds to both local and distal venous engorgement, and consequent stasis and diapedesis produce secondary ulcerative and parenchymatous inflammation in quick sequence.

Through the cervical nodes directly, and through lymphatic channels, salivary glands, ethmoid and sphenoidal sinuses more remotely, even the axillary spaces and pulmonary orifices are vulnerable.

Close relation to both cerebro-spinal and sympathetic nerve systems afford ready means for low grade neurotic irritation in the chronic inflammatory forms, and favor the severe algesia so distinctive of tonsillar and particularly peritonsillar involvement.

Again, a further important element in the consideration of tonsillar pathology, is the favorable focus the faucial tonsil presents

for aiding the absorption of the many products of intermediate metabolic change in the albuminoid metamorphosis.

Granting the possibilities of salivary absorption, and also the dangerous probability of cryptic stasis and accumulation of semi-toxic bacteral products, these degenerative changes may have a potent influence in producing the lethargic conditions of the body attendant upon tardy elimination. Absorption of toxins accidental or experimental has proven moderately rapid. Granting again the less constant presence in tissue interstices, fibrous sheaths and reticular spaces of such products as leucin, tyrosin, creatin, xanthin, etc, etc., and for a limited space remarkable exposure in this area, why should not this metabolic influence be consequential?

It is difficult to locate the cause of discomforting growing pains of early childhood, the low grade lethargic states of systemic function, frequent states of mental dullness and apathy, dull mottled skin. Is it not plausible to assume that free absorption of toxins through degenerated cryptal contents may have at least some influence in producing and maintaining these retrograde katabolic changes?

NO. V.

Comparative Changes in Chronic Hypertrophy.

It has been the writer's effort in outpatient service at the Children's Hospital, comparatively and statistically, to study the systemic and organic alterations accompanying this most common malady. Unfortunately at the present time sufficient data of value have not been obtained for statistical report.

The relief from pharyngeal obstruction, when such appears pathogenic, is marked and almost constant, but are there no fundamental grounds for judging hypertrophy and chronic lacunar tonsilar conditions, as essentially abnormal?

In consideration of this must be borne in mind the ready tendency to abrasion when hypertrophied, and consequent rapid absorption.

In connection with this paper the writer has been making comparative investigation of hypertrophic tonsillar changes at different age intervals. This in conjunction with Dr. Scarlett, of the University of Pennsylvania. Its purpose has been to ascertain if possible by sufficient ultimate averages the distinctive changes, particularly in parenchyma of the tonsil. Data obtained, however, have not in average presented sufficient distinctive characteristics

to warrant judgment. A number of slides have been obtained covering the marginal periods of normal adolescent growth, and a subsequent report will be forthcoming.

In general summary upon the all-important topic of tonsillar hypertrophy the following tentative conclusions will be accepted:

a. The tonsils become permanently hypertrophied as a result of single or repeated acute inflammatory changes.

b. These changes are both hyperplastic and hypertrophic in character.

c. The hypertrophy is most common between the ages of four and twelve, disappearing in atrophic change between thirty and thirty-five years of age.

d. The lymphoidal action gradually retrogrades upon occlusion of the eliminating surfaces and ducts.

e. Systemic absorption may be augmented by the atrophic thinning of the epithelial lining and decreased leukocytosis may accompany abrasion of the surface.

f. Whatever of functional importance may have attached to enlarged tonsils during periods of special lymphoidal activity, in all probability does not exist when such period has ceased.

g. Statistics do not indicate any systemic deprivation, either general or to special sense organs, after indicated surgical interference.

PAPER:

MODERN SURGERY OF THE FAUCIAL TONSILS.

ROBERT C. MYLES, M.D.

During the past ten years the writer has prepared and read several papers on the surgery of the tonsils before various scientific societies. In these articles he has described and advocated changes in the technique and instruments, as these changes have developed from his experience. A few specialists have seen fit to adopt these suggestions, because they have realized both their truth and importance. Many, however, have by omission refused to acknowledge the facts as presented, and it is to these confreres that I again make an earnest appeal.

The faucial tonsils, as I have observed them, may, for descriptive purposes, be divided into four classes, according to their relationship to the surrounding parts:

1. The Faucial Type, pedunculated in character, in which almost the entire tonsil is situated within the open space between the pillars of the fauces.

2. The Palato-lingual Type, in which one lobe of the gland is attached below and posteriorly near the tongue or posterior pillar, and the other lobe—usually about one-half—forms a palatal segment which extends upward, outward, and anteriorly from ¼ to ¾ of an inch within the faucial pillars.

3. The Embedded Type, in which at least three-fourths of the hypertrophied organ is embedded in the faucial and palatal walls.

4. The Inter-Mural Type, in which practically all of the tonsil is situated between the palatal and faucial walls.

I am aware that this classification is faulty and that many subdivisions could be made, but it may suffice as an aid in determining the method which secures the most effective removal. It has been said that true surgery deals with how, when, and where to cut. How to remove the diseased parts of the faucial tonsil is one of the most difficult problems in the realm of surgery. The simple removal of a pedunculated tonsil with a guillotine, a snare, or a knife, seems almost exclusively to have occupied the minds of many distinguished writers for the past half century. In the author's experience, only about 20% of all tonsils can be placed in the first, or pedunculated, class.

The cases which are classified in the second, third, and fourth divisions present conditions which test the surgeon's skill and efforts. It would seem that with an organ so easily accessible there should not be so much difficulty and obscurity about a proper diagnosis. The act of violent gagging places the palatal lobe of the tonsil in a most favorable position for determining any variation from its normal conditions. Firm pressure with a Frankel's tongue depressor at the external end of the base of the gland will usually express a part of whatever putrid or cheesy debris may be contained within the lacunæ or supra-tonsillar fossæ. The odor from its detritus should have much weight in the determination of operative procedures.

The most constant and most important sign that surgical interference with the tonsil is imperative is the enlargement of the

cervical glands. The indifference with which the general practitioner, the throat specialist, and the modern pediatrist have treated numberless cases of throat sepsis or toxaemia, evidenced by diseased conditions of the cervical lymphatic glands, has astonished me beyond power of expression.

There seems to be a difference of opinion in reference to the induction of general anaesthesia, and the application by injection or simple contact of local anaesthetics. The author employs both methods, preferring the nitrous oxide gas and ether for the cases requiring general anaesthesia; and cocaine swabbed thoroughly over and in the part of the tonsil to be excised. In adult cases, local anaesthesia is used almost exclusively. This is followed by a free injection of a one-eighth of 1% sterile solution of cocaine. This injection should be made in the submucosa and the connective tissue around the capsule of the tonsil. It is useless and deceptive to insert the needle in the tonsillar tissue, as the solution immediately passes out through the follicles. I present a special platinum needle made for me by Ermold. It will be noted that the needle is sharpened at almost a right angle to its long axis, in order that the thin covering of the tonsil may not be completely pierced.

Retching, coughing, vomiting and bleeding are some of the objectionable features that interrupt the technique of a tonsillotomy under local anaesthesia. In most cases of the first division, and in small tonsils of the other three divisions, the tonsils, including their capsules, may be practically enucleated when such a procedure is indicated; but the author has not considered it safe or feasible in classes II., III. and IV. to remove all of the capsule or fibrous investment sheath in cases where there is extensive development of the lymphoid tissue within the faucial and palatal walls.

After the tonsillar region has been anaesthetized, as described above, the tonsil is separated from its capsule as well as the conditions will permit. The automatic scissors which I present, and which were made by Ermold under instructions from myself and my assistant, Dr. A. M. Anderson, is a useful instrument for this purpose. The scissors are made after the alligator forceps pattern, with a beveled lever to force the blades together during the act of cutting. I also use some of the tonsil or scalpel knives shown in connection with this paper.

When the capsule which covers the anterior portion of the tonsil posterior to the free border of the palate-glossus muscle projects prominently and forms the involucral or opercular fold of Harrison Allen, the writer usually removes this part of the capsular sheath, which is composed mostly of mucous membrane. The central tonsil mass is seized with dull forceps or tenaculum and drawn through a very small Mackenzie tonsillitome or wire snare loop, and severed. In many cases it is better not to use the traction forceps, as the adjustment can be better made with side pressure on the tonsillotome or wire snare. This step is followed by the use of the author's punch forceps, preference being given to the one with the cutting blades placed at an obtuse angle to the shaft. The cutting should be done upward, outward and forward. In only a limited number of adult cases can the capsule be inverted, and when held in that position the irregular tonsil masses can be removed safely with the forceps.

The construction of the punch or double-scissor forceps makes them more or less automatically safe in intelligent hands when cutting in the deeper recesses of the capsular sheath. The distal ends are blunt, and one blade is necessarily longer than the other, making it difficult to engage any part but the soft irregular tissue. It is best, when possible, to draw the mucous membrane and anterior pillar forward with a very delicate tenaculum and remove under direct inspection with the small forceps the various lymphoid masses that may be attached to the sides and the bottom of the sheath cavity.

In reference to remvoal of the capsular sheath, as advocated in all cases by some recent writers, I will state that I have not been able to follow their technique in cases of deep development coming under classes II., III. and IV., without at times causing, in my judgment, unjustifiably severe reactionary symptoms—such as pain for several days, sepsis, fever, edema, cellulitis, extending in some cases to the external neck and zygomatic regions. I have noticed that the unfavorable reaction after removal of the tonsils or parts thereof has been proportionate to the wounding of the adjacent tissues, and that very little pain, inflammation, or bleeding are caused when the cutting is restricted approximately to the tonsil tissue alone.

When anaesthesia by gas or ether is used in cases of children, the child is placed on a table, by preference 36 to 40 inches high.

It is better to secure profound anaesthesia before commencing the operation, in order that one may, in many cases after a single administration, be able to remove both tonsils, including the palatal lobes, and also have plenty of time to thoroughly remove the adenoids with forceps guided by the finger, and for the after use of the curette. In order not to lose any of this important time, the writer has worked out and adopted the following plan and technique in all cases of the II., III. and IV. classes while the patient is lying on his back and after the introduction of the mouth gag.

The base of the tongue is depressed downward and forward; then one blade of the automatic scissors is placed in the supra-tonsillar fossa or an upper lacunæ, and the other in the faucial space a few millimetres posterior to the anterior pillar, and section is made downward through the mucous membrane and the capsular sheath covering the anterior surface of this segment. This procedure is especially useful in the II. and III. classes, and only occasionally necessary in the I. and IV. The tonsil is then separated from its superior and posterior sheath wall, with straight or curved scissors and knives, as carefully and as well as may be possible without using too much time; if the haemorrhage is not annoying and the patient is still lying on his back, the small guillotine or the wire loop is carefully adjusted over the central mass and the portion engaged is removed. The patient is now turned over until he lies on the anterior part of the under hip, the head is placed over the edge of the table, and in a downward position directed so that the blood will flow out of the mouth into a basin. It is my custom not to stop operating on account of the bleeding, but to proceed with the punch forceps, excise the basis, or palatal lobe, of the tonsil on the upper side, and allow the blood to flow across the tongue and out of the most dependent corner of the mouth. Many years ago an accident revealed to me one of the most important points in the technique. When trying to remove some tissue from the bottom of a capsular sheath, the forceps were too dull to cut away some fibrous tissue, and firm traction was made hoping to tear the tissue away. Suddenly the capsule became inverted, and a large mass of cauliflower-like appearance protruded upon the base of the tongue at a time when I thought that I had already performed an almost complete tonsillotomy. Since then it has been my custom, in cases of children, after removing the central part of the base of the tonsil, to

invert the capsule after the manner described above, and hold it in this position with one hand, while with the other I remove practically all of the lymphoid tissue which had previously been situated so deeply within the walls of the fauces. The resultant cavity subsequently has the appearance of a crater, but it gradually becomes smaller by contraction of the capsule. The patient is then turned on the other side and the same technique is carried out for the other tonsil. After the patient is turned on his side, the author operates in the kneeling position, using a small cushion to protect the knees from the floor. He also uses an electric head light, supplied by a small battery in the breast pocket of the operating gown.

In cases of haemorrhage, either immediate or secondary, a small layer of cotton is placed on the tip of the palmar surface of the index finger of the same side as the bleeding tonsil. The cotton is covered with a paste of tanno-gallic acid, and is held on the bleeding point for five to fifteen minutes. If this does not arrest the bleeding, an artery forceps is applied, a fine catgut suture is passed through the tissue at the point of bleeding and tightly tied.

The writer is aware that a paper of this length cannot be complete, but hopes that much will be filled in by the discussion. He believes that the profession has done its duty in the cases of the first division, but that we have been chiefly masquerading as tonsillotomists and tonsillectomists in the classes of the II., III. and IV. divisions. It has been noted that occasionally in very young children an amount of tonsil tissue will recur or grow again, which requires a second removal, even after what seemed a thorough operation, but this does not now occur once to where it did twenty times after the old method of using the guillotine only.

PAPER:

MODERN SURGERY OF THE FAUCIAL TONSIL.

THOS. J. GALLAHER, A. M., M. D.

As the success of any surgical procedure is dependent upon an accurate knowledge of the anatomy of the part, I beg the indulgence of this society while I briefly describe the surgical anatomy of the tonsil and its surroundings.

The sinus tonsillaris is a triangular area on the lateral wall of the pharynx, bounded anteriorly by the anterior palatine arch; posteriorly by the posterior palatine arch; externally by the superior constrictor of the pharynx; and below by the side of the pharyngeal portion of the tongue; above it is limited by the junction of the two arches. It contains the tonsil.

At the junction of the two arches, a reduplication of the membrane is found, the plica triangularis, which covers the supra-tonsillar fossa. As the membrane covering the anterior and inferior portions of the tonsil is also called the plica triangularis, some authors call this plica above the tonsil, the plica supra-tonsillaris, which avoids confusion. The plica supra-tonsillaris, hence, bounds the supra-tonsillar fossa above with its semilunar margin. It has an anterior and posterior cornu which are continuous respectively with the anterior and posterior pillars. The plica tonsillaris inferior is reflected from the mucous membrane of the anterior pillar upon the tonsil, covering it anteriorly and extending along its inferior border for a variable distance, often reaching the posterior pillar. At the point where the plica is reflected upon the tonsil, a sharp angle is formed by the plica and the anterior pillar, which may properly be called the commissure of the anterior tonsillar fossa.

The tonsil, together with the different reflections of the mucous membrane, forms with the arches, three distinct fossae, viz., the anterior, supra-tonsillar and posterior.

The anterior fossa is bounded in front by part of the anterior pillar; above by the commissure of the anterior fossa (or point of

divergence of the anterior palatine arch and plica triangularis) ; posteriorly by the anterior border of the tonsil (with its plica) ; below by the tissues of the side of the tongue. The supra-tonsillar fossa is bounded above by the semilunar margin of the plica supra-tonsillaris ; anteriorly and posteriorly by the anterior and posterior cornua of the plica supra-tonsillaris ; below by the superior margin of the tonsil. The posterior fossa is bounded anteriorly by the posterior border of the tonsil ; above by the commissure, that is, the point of divergence of the mucous membrane of the posterior pillar and the membrane reflected from it upon the tonsil ; posteriorly by the posterior arch ; inferiorly it is continuous with the lateral wall of the pharynx. The anterior fossa is always present and is usually of considerable size. The superior fossa is also constant and quite variable in its extent. The posterior fossa is not so distinct, but can usually be made out. The anterior and posterior pillars are formed by the projection of the palato-glossi and palato-pharyngei muscles covered with mucous membrane. The posterior pillar on each side is nearer to its opposite arch than is the anterior to its opposite, consequently the posterior pillar covers much more of the tonsil than does the anterior. It is owing to this fact that the posterior fossa is limited in its extent.

The disribution and direction of the tonsillar crypts perhaps influence us most in determining the extent of the tonsil operations. The crypts in the superior portion extend downward and outward to the capsule and are often closely covered by the plica supra-tonsillaris. The crypts in the middle and lower portions of the tonsil extend more horizontally and upward. The middle and lower crypts are, therefore, more easily drained than the upper crypts. It is from the upper crypts usually that infection produces peri-tonsillar abcess. It is of greatest importance that the velar or superior lobe of the tonsil should be removed to prevent a recurrence of this infection. The crypts are both simple and compound and extend from the surface of the tonsil externally to the capsule. The capsule is the fibrous membrane covering the external portion of the tonsil and is loosely attached to the superior constrictor.

We shall briefly call attention to the blood supply of the tonsil. The arteries supplying the tonsils are the dorsalis linguae from the lingual, the ascending palatine and tonsillar from the facial, the ascending pharyngeal from the external carotid, the descending

palatine branch of the internal maxillary and a twig from the small meningeal.

The point of practical importance in regard to the blood supply is that the arteries pass external to the superior constrictor, through which they continue and then divide into the smaller vessels, which enter the tonsil through the capsule, hence care should be exercised that none of the vessels be wounded far from the capsule. Normally, the internal carotid artery lies behind and to the outer side of the tonsil nearly an inch distant from it, in the posterior portion of the pharyngo-maxillary interspace. Barring an anomalous position of the internal carotid, which will be shown by the visible pulsations of the tonsil, this vessel is not at all likely to be wounded. The veins terminate in the tonsillar plexus in the outer and lower portion.

A branch from the glosso-pharyngeal nerve, by uniting with the branches of the pharyngeal plexus, forms the tonsillar plexus.

The excellent work of Goodale, Wright, Ballenger, Myles, Mosher, Wood, Robertson, Babbitt and others has given the proper status to the normal and diseased tonsil. There is no longer any doubt but that many infectious germs gain entrance to the system through the tonsil. It is, indeed, a sign of the times that the so-called rheumatic attacks following acute tonsillitis have been placed in the category of septic infections.

Operations upon the tonsil may be divided into the simple type of operations and the radical tonsil operation. Among the simple operations may be included the slitting of the crypts by the knife or galvano-cautery, reduction of the tonsillar tissue by the galvano-cautery, and lastly tonsillotomy. These operations are seldom of choice and are usually done only when it seems most expedient under existing circumstances. As an example, it is better to reduce a hypertrophied tonsil by means of the galvano-cautery rather than do the radical operation when any doubt exists as to the position of the internal carotid or any large anomalous vessels. As to tonsillotomy, it is certainly a most incomplete operation and its results are very often most disappointing. The tonsillotome soon will be seldom used.

The radical tonsil operation consists in the complete exenteration of the sinus tonsillaris. As radical operations are rather in favor today, I take the liberty of using the term in reference to the tonsil. There is no more reason for doing an incomplete tonsillar operation,

when its removal is indicated, than there is for imperfectly performing any surgical operation. While many operators have been very careful to remove all of the diseased mastoid cells, yet they but partially remove the diseased tonsillar tissue.

We will not discuss the indication for removal of the tonsil, but when its evisceration is attempted the radical operation only should be done. It is only such work that will prove satisfactory to both the sufferer and operator. We can assure our patients perfect immunity from tonsillitis, peri-tonsillar abscess and the serious constitutional disturbances. Indeed, the failure to bring the desired relief by the partial removal of the tonsil has brought upon the laryngologist much just criticism. In the institutions in our larger cities today, it is still an exception to find an operator who is doing a complete radical operation. Great credit should be given to Ballenger, who has been advocating the complete removal of the tonsil for years. The first complete enucleation which I saw was performed by him some years ago. Since coming to this meeting he tells me that he has still further improved his technique, which I hope he will describe in his discussion before the society.

Every operator will, of course, have his favorite method and many techniques have been given to the profession within the last few years. A large number of instruments have been devised for the purpose, including knives, scissors, various punches, snares, et cetera.

In performing the radical operation, personally, I use the following: A suitable tenaculum forceps, the Pierce knife, the Kyle crypt knife, which has long been recommended by Ballenger, and a slight modification of the Peters' snare. The operation may be called the dissection and snare operation. The snare is often improperly used and should never be placed around the tonsil without a thorough loosening of the organ from the sinus tonsillaris. It matters not about the shape of the tonsil or as to the amount of tonsillar tissue projecting beyond the pillars. This operation will always be found applicable and thorough. In the adult, when operating without general anesthesia, we inject a mild solution of cocaine and adnephrin or adrenalin along the line of incision, waiting one minute and proceeding. In children under general anesthesia, occasionally we inject a weak adnephrin or adrenalin solution or apply locally.

The tonsil is first seized with tenaculum forceps and drawn internally to accurately show its outline. An incision is then made

with the Pierce knife along its posterior border, which quickly and easily frees it from its attachment. This incision extends from the lower margin of the tonsil along its posterior border to the commissure of the posterior fossa. The Kyle knife is then used, cutting through the posterior commissure, and extending the incision over the upper margin of the tonsil,—that is, through the supra-tonsillar fossa. Next an incision is made with the Kyle knife along the inferior attachment of the tonsil and extending upward, separating the plica triangularis inferior along its outer boundary to the commissure. This is continued upward through the commissure and joins the upper incision in the supra-tonsillar fossa. After these incisions are made, the tonsil is no longer held within the sinus tonsillaris and a slight traction on the forceps, toward the median line, will cause a beautiful eversion of the tonsil, so that the capsule of the tonsil which is loosely attached to the constrictor is drawn internally to about the same plain as the anterior pillar. The dissection can then be continued directly backward, and also, further liberation of the tonsil made along its superior margin. The entire tonsil can be dissected out by means of these knives, but the use of the snare facilitates the operation somewhat and possibly lessens the danger of hemorrhage. We use the No. 7 piano wire and a slightly modified Peters' snare, with no tips, and find that the plain wire can be much more easily insinuated external to the capsule. The same wire can be used for both tonsils. The tonsil is usually removed completely with its capsule intact. After the operation, the sinus tonsillaris should be carefully inspected with a small forceps to see if any fragments of the tonsil remain, especially in the upper portion. If any are found they should be removed.

Dangerous and Faulty Technique: The plica supra-tonsillaris should only be cut through at the anterior and posterior cornua and not through the semilunar margin.

Laceration of the constrictor and wounding of the larger vessels external to it which would produce very troublesome hemorrhage.

Cutting of the palato-glossi or palato-pharyngei muscles which will certainly give contractions of the palate. The contractions are more liable to be produced by the laceration of the posterior pillar.

Incomplete severing of the marginal attachments which permits the surrounding tissue, especially above, to be drawn into the snare and wounded.

Insufficient loosening of the capsule from the constrictor.

Too much stress cannot be placed upon the importance of the careful dissection of the velar lobe from its attachments, as it is common for the inexperienced operator to leave much of this lobe in the sinus. Hence, it is wise to always inspect the sinus as suggested above.

In a record of over one hundred and twenty-five cases, in the adult, by this method, the only bleeding that we have had has been incidental to the dissection, and in each instance, after the snare had completely enucleated the tonsil, the bleeding stopped spontaneously. My experience has been that it is in the cases of incomplete removal of the tonsil that the serious hemorrhages occur, the soft tissues preventing proper retraction of the vessel. In children I prefer general anaesthesia.

The technique as described above requires simple and few instruments and certainly insures a most thorough evisceration.

While the radical tonsil operation requires much care and a keen appreciation of the anatomy, I am satisfied that when any operator has become proficient in its performance that there will be but one operation which he will do for those suffering from diseased tonsils —the one which will positively insure immunity from any further inconvenience—the one which will be a boon to his patient and a satisfaction to himself,—namely, the Radical Tonsil Operation.

DISCUSSION.

Dr. William L. Ballenger, of Chicago, held that complete removal of the tonsil would obviate any further trouble for the patient, except such as that mentioned by Dr. Myles. He thought there had been a needless invention of instruments and an unnecessary expenditure of money for instruments for the removal of tonsils. With a pocket knife and good grasping forceps he could remove any tonsil in adults with its capsule intact in a very short time. An ordinary sharp scalpel and forceps of the vulsellum type were the only instruments he used in removing tonsils from adults. He operated under local anesthesia by the injection method mentioned by Dr. Gallaher. With a few deft movements of the knife the tonsil could be completely removed, with its capsule intact, with the best healing wound it was possible to have. He had found that hemorrhage was in proportion to the wounding of vessels in the muscles of the pillars. There should be no muscular tissue removed. He described in detail his method of procedure.

Dr. Thomas J. Harris, of New York City, hoped that the papers presented and the discussion thereof would settle the various

questions which had long been doubtful concerning the surgery of the tonsil. The subject of the technique could very properly be allowed to rest, but he would be very sorry if the question of con- in children under five years of age he believed to be of this class. servatism versus the radical side were allowed to pass. The question might justly be asked whether the radical operation is always required and whether it will do what it is desired to do.

Dr. Harris P. Mosher, of Boston, had not found the anatomy as it had been outlined by the previous speakers. He had never been able to make out the division of the supra-tonsillar fossa into an upper and a lower. It had always seemed to him that the pharyngeal tonsil had rather the better of the faucial tonsil in the matter of drainage. In his study of the subject he had found the two to be situated more nearly on a level than was usually supposed. The part of the faucial tonsil which did not drain well was that covered by the plica. He hoped he would hear no more of the internal carotid artery in connection with the tonsil—there was bleeding, but not from the internal carotid. He was glad to hear Dr. Ballenger advocate taking as much time as was necessary for the operation. He saw no necessity for such great speed. He al- l, in which the tonsil is not embedded. Ninety per cent of cases ways took off the plica flush with the anterior pillar.

Dr. John F. Barnhill, of Indianapolis, Ind., had always rather He had had the opportunity of watching these children as long as favored radicalism in the surgery of the faucial tonsil, with the exception of the class of cases to which Dr. Myles referred as No. ten years after operation with the guillotine, and he had seen no recurrence and no quinsy. The important thing at this early age was the removal of the obstruction to breathing, and in ninety per cent of such cases the simple operation was all that was necessary. Where the tonsil was embedded, however, the simple operation would not suffice. He had always been satisfied if he could remove all tonsillar tissue beyond the bottom of the crypts.

Dr. George B. Wood, of Philadelphia, referring to what Dr. Babbitt called chronic tonsillitis, said that he considered this term a misnomer. The condition was an accumulation of desquamated epithelial cells in the crypts of the tonsils. This mass of cells being retained in the tonsil crypts became infected and underwent decomposition. The treatment should consist in thoroughly opening up the crypts as it was not necessary in these cases to completely remove the whole tonsil. In this condition of lacunar retention the tonsil was not necessarily inflamed, though it might be so coincidentally. Dr. Myles's tonsil punch was an excellent instrument for removing either a portion or all of the tonsil, but it could be greatly improved by having the cutting blade sharpened to a knife edge. A sharp cutting instrument was always much less painful than a blunt instrument like the ordinary punch. He urged that the members drop the term "adhesions of the tonsils." With the

exception of abscess perforating through the anterior pillar he had never seen adhesions other than those of congenital origin.

Dr. H. W. Loeb, of Saint Louis, said it was not at all uncommon to see cases of tonsillitis of the variety referred to by Dr. Myles as No. 1 give an immense amount of trouble, and to see cases of the variety known as No. 4 give absolutely no symptoms. It was a question whether one should anticipate trouble. The radical removal of the tonsil should depend upon the degree of the symptoms. Where the symptoms were not particularly important, where the patient was not subject to much pain by recurring inflammations, it was best to leave the tonsil. On the contrary, where a very small part of the tonsillar tissue remains, or where the variety No. 4 causes trouble, removal should be undertaken. Tonsils which have been snipped off by the general practitioner have been seen to go down and to give no further trouble. Each operator must be guided by the personal equation, both on his own part and on that of the patient.

Dr. John B. Roberts, of Philadelphia, held that the position of the patient simplified the operation materially, the upright position being that in which it was easiest to reach the tonsil and to control the bleedng. He had frequently operated with the patient in the sitting position, and without general or even local anesthesia. The sensation to pain in tonsillar tissue was very little developed.

Dr. Clement F. Theisen, of Albany, N. Y., did not agree with the statement that complete removal of the tonsil would obviate any further trouble. The patient would not have tonsillitis again, but he might have inflammation of the surrounding tissues which would give almost as much trouble as tonsillitis. He took issue with Dr. Gallaher concerning the tonsillotome, holding that in some cases the tonsil could be practically removed with this instrument. He used the tonsillotome first, then completed the operation with the tonsil punch.

Dr. Frank B. Sprague, of Providence, R. I., referred to the fact that within the past fifteen years twenty deaths had been reported from chloroform in adenoid and tonsil operations, and urged caution in the use of this anesthetic. He had found it easy in children to remove the tonsil completely with the guillotine tonsillotome, by placing the opening over the tonsil from behind, pushing back the plica and the pillars. Should there be a second lobe this could be removed by reaching down beneath it and pressing upward and forward, and any remaining pieces could be removed by the Myles or Farlow punch. He had had severe hemorrhage to occur after the fifth day subsequent to operation. He cited a case of primary hemorrhage in a young woman of eighteen who had formerly had a cyst of the tonsil half an inch in diameter. A few weeks later he had operated for removal of the adenoids and tonsils, clearing away everything but a small tab of tonsillar tissue, which he clipped off with the punch. As he did this the blood spurted out. He placed

his finger on the bleeding point, then applied a pad of cotton soaked in glycerite of tannin, allowing this to remain *in situ* for five days. There was no further bleeding and all went well with the case. Removal of the tonsil had been advocated to relieve difficult breathing; he had seen enormous tonsils with very slight interference with breathing. He believed that large tonsils were less obstructive to breathing than adenoids. Almost invariably, where there was hypertrophy of the tonsils, the adenoids were also enlarged, and removal of tonsils without removal of adenoids was useless.

Professor Killian, of Freiburg, Germany, said that visitors to his clinic would see that he and his associates were not radical. He gave no general anesthetic as a rule, and if he did give it it was only for a moment. He had not found it necessary to employ general anesthesia, as the operation is not painful and is a very short one. He used the tonsillotome as a rule. It was not necessary to remove all the tonsillar tissue or all the adenoid tissue in the vault of the pharynx. The adenoid tissue has a very important function in the production of saliva and for protection against infectious disease. The tonsils manufacture lymphoid cells and aid in digestion. The function of the tonsil in the naso-pharynx was not established. He believed, however, that it served some purpose. There should be some indication for the radical operation. If a child had tonsillitis every two weeks this should be considered an indication for radical removal; if it had one or two attacks a year it was not necessary to remove the entire tonsil. In the latter case it was only necessary to remove such tissue as was above the normal size of the tonsil. He had observed, in cases where he had operated radically, if the child had a small amount of adenoid tissue this was prone to become inflamed, and it was necessary in all cases to give local treatment. Should the child be unable to breathe through the nose not only the nasopharynx but the nose as well should be cleared out.

Dr. Christian R. Holmes, of Cincinnati, asked the readers of the papers to say, in closing the discussion, whether patients complained of pain after the radical operation. He had found that some of them complained of pain for two or three days after the use of the snare. With reference to the question of anticipating trouble, he said it had been his custom, in giving an anesthetic for the removal of adenoids, to always investigate the tonsils and especially the supra-tonsillar space. He had used chloroform for a number of years, but had at last become afraid of it, and now uses ether or ether in combination with nitrous oxid.

Dr. Myles, in closing the discussion, said it was taken for granted that when the radical operation was advocated the indications were sufficient to warrant this procedure. He had tried to emphasize in the paper that enlarged cervical glands give positive evidence of tonsillar sepsis. He could not understand why pediatrists had not given more attention to and kept more careful records with refer-

ence to enlargement of these glands. Instances which were formerly attributed to scrofula he believed to be due not to scrofula but to tonsillar sepsis. In general rheumatism, myocarditis and other affections associated with tonsillitis, if the finger were passed along the borders of the sterno-cleido-mastoid muscle, these glands would be detected. If this evidence of tonsillar infection were always looked for, and if the tonsils in such case were always removed, the visits of the general practitioner would be cut down twenty per cent. He spoke of the proximity of the pneumogastric nerve to the very large inter-mural tonsil, and took issue with Dr. Mosher concerning the matter of the probability of injury to the internal carotid artery, which passes just behind this class of tonsil. The reaction following operation was greater in children than in adults, and was in proportion to the wounding of the peritonsillar tissue. Could the cutting be confined to the tonsillar tissue there would be but little reaction. It was always his practice to leave some part of the sheath in the deeply embedded type of tonsil. The pathology of the crypts and lacunae was being carefully studied. The detritus in the crypts he believed to be epithelial debris rather than food particles.

Dr. Gallaher, in closing the discussion, emphasized the fact that the tonsil is a superficial lymphatic gland, with direct communication with the lymphatic glands of the neck, and that it might be the seat of the primary infection in tuberculosis. Before condemning the radical operation it would be necessary to understand the physiology of the tonsil better than it is understood at the present time. Hemorrhage should be controlled in operations on the tonsils in accordance with ordinary surgical procedure. The snare was only an incidental part of the operation. With the dissection the operation could be completed with the snare in a second. It was then his custom to blow in some orthoform, and to direct the patient to do this every hour or so. He had done 150 radical operations and had not had a single peritonsillar abscess.

PAPER:

THE SUPPURATING MIDDLE EAR DISEASES OF INFANCY AND CHILDHOOD.

S. J. KOPETZKY, M.D.

It is a familiar fact that a completely developed organ is less liable to disease than a growing one with its proliferation of cellular elements and abundant blood supply. Being one of those regions of the human body, immature at birth, possessing a developmental stage extending through the early years of life, only reaching maturity in many instances with the advent of puberty, the juvenile ear possesses inherent elements which readily make it a field for disease.

Searching through the literature, one finds, in contradistinction to the case in adults where symptomatology and diagnosis preceded pathology, in the case of children the richness of autopsy material caused many pathological facts to be uncovered long before diagnosis, exercised during the patient's life, appreciated their existence. This is understandable when the difficulties attending even the cursory examination of the child are taken into account. Furthermore, the absence of subjective or functional examinations, because of what must be apparent—their absolute impracticability in many instances—class the suppurative ear disease of infancy and childhood among those whose fatal termination often alone bring evidence of their having existed; and, where not fatal, whose sequalæ in handicapping the later man or woman in their after career, by varying degrees of deafness, best point the gravity of their attack.

Hence this study of the suppurative ear diseases of childhood, to place the work of competent clinical observers and laboratory workers together, in such form that a better understanding of this prevalent condition may result.* I have busied myself with this study for some time, and in the following, where deemed advisable, I

*I have not attempted to quote the entire literature, limiting myself as far as possible to those whom I have been able to find as the original reporters on the given point, or where controversial questions would seem to require authoritative substantiation. Cumulative authority has been eliminated as far as possible, hence many well known names will be found omitted.

have added personal cases, and also the results of anatomical studies wherever data thus obtained conflicted with or emphasized the writings of others. I have limited myself to middle ear suppurations, merely touching upon some of its sequalæ, because even at that the limits of this paper have gone beyond my original intentions.

We are indebted to Gruber,[1] to Haug,[2] and to Gomperz,[3] among others, for suggesting the possibility and feasibility of examining the external auditory canal and the drum in even the youngest child, so that today, at least, the attempt at such examination is usually made, where formerly diagnosis in these cases rested on facts obtained per exclusion. I believe from personal experience that in at least 80% of all children a view of the membrana tympani is obtainable.

I. PREVALENCE.

A glance at the published statistics will convince one of the prevalence of this condition. Thus, after examining 22,894 school children in the city of Zurik, Laubi[4] found that 14% of all children under 4 years of age were affected, the most frequent anomolie being some form of tubal trouble.

The frequency with which various pathologic conditions were demonstrable was as follows:

Tubal trouble	51.1%
Remains of Otitis Media Purulenta	16.2%
Internal Ear involvement	14.4%
Cerumen	6.5%
Otitis Media Chron. Catarrhal	5.4%
Otitis Media Purulenta Chronica	2.4%

Barschmann[5] examined children to determine the hearing faculty with the following results; he divided his cases into five groups, viz.:

1st group could not distinguish acumeter or voice beyond 5 meters.	
Result: For acumeter	3.0%
For voice	2.3%
2nd group could not hear acumter beyond 6-8 meters	5.5%
2nd group could not hear voice beyond 6-8 meters	3.8%
3rd group could not hear acumeter beyond 9-10 meters	6.6%
3rd group could not hear voice beyond 9-10 meters	5.4%

4th group could not hear acumeter beyond 11.15 meters..... 9.0%
4th group could not hear voice beyond 11.15 meters.....21.8%
5th group had normal hearing.

Of those examined, he found that as age increased larger numbers were hard of hearing. His results further demonstrated: Pathological changes in drum and external auditory canal in 3% of the cases, scars in the drum were present 14.4%, the retracted drum was evident in 14.2%, while 5.3% showed perforated drums, some having, and others having had, aural discharge. He found the defectiveness of hearing in direct proportion to the demonstrable pathological changes present.

Fig. 1. Fronto-vertical section through the external auditory canal, the drum and the pyramids of a year old child, showing the direction of the canal, its attachment to the S. petrosus meatus and the nearness of the middle ear to the surface of the skull. The thin wall of bone separating the tympanic cavity from the jugular bulb is also indicated. (Drawing partly schematic, after Gompers.)

In order to estimate the frequency of occurrence of middle ear suppuration, as compared with adults, it is only necessary to glance at the reports of Burkner and Bezold and at my own findings.

Of all Burkner's patients with ear suppurations (both acute and chronic), 36% were adults and 64% children. Bezold, on the other hand, reports of his patients that 20% were children and 80% were adults. The difference in these two reports lies in the fact that Burkner's report is restricted to suppurative conditions, while Bezold's refers to all ear diseases.

Through the courtesy of my chief, Dr. Wendell C. Phillips, I instituted an examination of the records of our clinic at the Manhattan Eye, Ear and Throat Hospital, New York, and find that for the last seven years and two months there were in all 7,300 cases of ear disease recorded. Of these 2,116 were children ranging in age from a few days to 18 years. That is, of all the cases treated 28% were children.

The following is the tabulated result and percentage of frequency of the diseases found to have existed in the children at the time they applied to the clinic for treatment:

Diseases.	Number of cases.	Percentage of Frequency.
Otitis Media Purulenta Acuta.....	710	33.0% +
Otitis Media Purulenta Chronica.....	696	32.0% +
Otitis Media Catarrhalis Chronica....	281	13.0% +
Mastoiditis	97	4.0% +
Furunculosis	25	1.0% +
Deafmutism	27	1.0% +
Labyrinthine trouble	5	0.2% +
Cerumen	137	6.0% +
Sundries	128	6.0% +

From this table it is apparent that 69% + of all the children applying for treatment suffered from middle ear suppuration, the mastoid being involved at the time of admission to the clinic about 4% of times. During the first year of life Barth[7] places the percentage of all children having otitis media as high as 80%, his observation being based on the results obtained from 600 cases and autopsies.

Furthermore, in material coming to autopsy from children whose death had been ascribed to various causes, the post-mortem examination demonstrates that the ear is diseased much oftener than is suspected. Thus, Ponfick[8] found in 100 autopsies, where death had been ascribed to various causes, that only 9 cases evidenced normal ears. These cases were from the Breslau Klinik, with ages varying from one day to 4 years. His results showed both ears involved 78 times, and unilateral involvement 13 times. He reports that his post-mortems demonstrated, aside from a moderate enlargement of the spleen, that the ear alone showed pathological changes, and he concludes that otitis media may occur as an independent infectious disease capable of causing death.

Not only are many children affected, but many of the ear diseases of adult life date their origin to the early days of life. Of 100 cases of chronic middle ear suppuration among adults, Barbarin[10] found that 42 were traceable to the days of infancy or childhood. I found about 27% of the adult cases giving histories which lead me to hold that the ear trouble in these was the sequala of an active process during the early formative period of the ear's development.

II. CAUSES OF PREVALENCY.

Having established their prevalency, what are the causes underlying the suppurative middle ear diseases of infancy and childhood which tend to produce this prevalency?

These underlying factors may be classified as follows:

1. General constitutional disease, and the hereditary influence of tuberculosis and syphilis.
2. The prevalence of the exanthemata during childhood.
3. Anatomical structures peculiar to childhood in the ear and its annexa.

1. Regarding the general diseases whose presence in childhood predisposes toward the prevalence of otitis media, it may be said that any general disease which tends to weaken body resistance lays the child liable to ear disease. Among such must be recognized rhachitis, bronchopneumonia, and the gastro-intestinal derangements. (Rasch,[11] Kassel,[12] Hartman[13].)

Regarding the latter, I differentiate between gastro-intestinal disturbances per se and those bowel and stomach derangements produced by the otitis media, of which I will speak hereinafter.

Furthermore, a child afflicted with tuberculosis or syphilis is very apt to develop middle ear disease, which may or may not be of itself tubercular or syphilitic in its pathological findings. Thus, Henrici[14] concludes that one-fifth of all cases of infantile mastoiditis is due to tuberculosis, and, although Barbarin[10] does not believe tuberculosis responsible for the many infantile otitis medias, still he reports 10% of 100 cases of chronic suppurations presenting tuberculous caries. (Haike,[15] also noting its existence, believes its extension from the pharynx through the Eustachian tube in contradistinction to the views held by Henrici, who believes the circulation responsible for its presence in the ear.)

But it is especially of tuberculosis or syphilis in the parents to which I allude as being distinctly of moment when considering the

causes underlying the prevalency of ear diseases in childhood. Körner[16] holds that one of the predisposing factors are tuberculosis or syphilis in the parents, the children themselves not necessarily being either tuberculosis or syphilitic, especially holding it of importance when advanced tuberculosis is present in the father at the time of procreation. In this connection it is of interest to note the report of Ostmann,[17] who, basing his results upon the examination of school children in Marburg (Germany), determined that those families which included the relatively highest numbers of children with hardness of hearing, likewise presented the relatively most frequent taint of tuberculosis heredity.

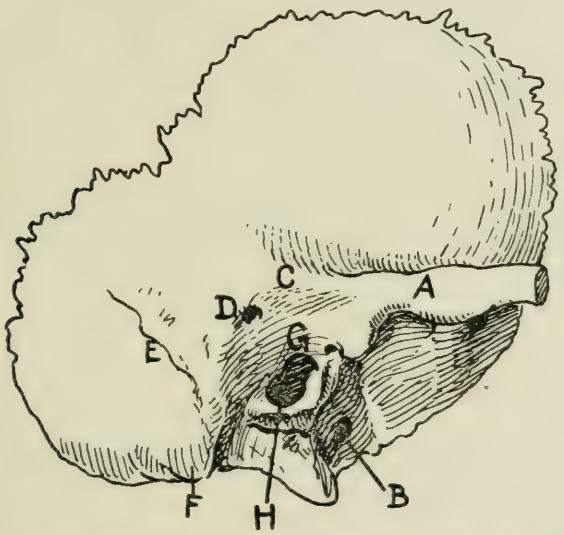


Fig. 2. Outline drawing of the temporal bone of a two-and-a-half-year-old child, showing landmarks. A. Zygomatic process. B. A defect in the ossification of the anterior inferior bony wall of the external auditory canal. C. Supremastoidal crest. D. Point of election for entering the antrum. E. Squamo-mastoidal suture. G. Superficies meatus. H. Osseous opening of the external auditory canal.

I have observed, in taking histories of underdeveloped children, with recurrent swellings of the cervical glands, that their parents, while often not admitting being tuberculous, at least give signs by their general appearance which would lead to a suspicion of its presence—a suspicion unfortuntely which cannot be proved. Again it has been my experience that these cases, when operated upon, and with no mistakes as to technique, still do not get well, and I have always felt that the constitutional tuberculous taint, which I suspected but could not prove, was the cause of the trouble.

2. The exanthemata, contagious as is their nature, attacking many children and bringing in their train the involvement of the ears in many cases, are thus in themselves important factors influencing the prevalency of ear disease in childhood.

Downie's report cited by Holt[18] shows 501 cases of middle ear suppuration following the exanthemata; he reports that measles preceded the ear disease in 26%, scarlet fever in 12%, and whooping cough in 15% of the cases. Bezold[19] and Jansen[20] estimate that 5-10% of all ear disease is due to scarlet fever, and at least from 12-28% of all purulent ears are its sequalæ according to Haug.[21] Heubner,[22] studying 393 cases, reported 27.4% ear complications (scarlatina), and Burchardt-Merian,[23] describing an epidemic of scarlatina, observed the ear involved in from 22 to 33%.

It is to be noted in passing, that among the initial symptoms of scarlatina there is presented a swelling of the adenoid tissue of the pharynx.

While formerly it was held that measles did not affect the ears as readily as does scarlatina, more recently it has been demonstrated that ear complications follow measles almost as often as they do scarlatina.

Wendt[24] places ear complications following variola as high as 98%, but it must be borne in mind that this disease is not very prevalent with us, and hence plays small role as a factor determining prevalency of ear suppurations in childhood.

Epidemic influenza, mumps, and pneumonia complete our list of diseases, rather prevalent in childhood, whose evil sequalæ are presented in the form of ear complications.

3. Among the anatomical structures peculiar to infancy and childhood which are factors tending to cause prevalence of ear disease at this age, we find that the Lymphatic Ring, of Waldeyer, takes an important place, and of this lymphatic ring the adenoid is of greatest importance.

The adenoid of childhood is in itself not to be considered as an etiological factor. On the other hand its diseased condition is.

The presence of adenoid tissue in the child's naso-pharynx is, in my opinion, a normal physiological growth. The peculiarities of the infant's Eustachian tube will be reverted to later. At this time I wish merely to allude to its shortness of length, its widely open lumen, and its horizontal direction. Bearing these things in mind, it has always seemed to me, aside from whatever other

physiological function the adenoid may have to perform in the body economy, that nature used the adenoid to block the wide-open pharyngeal orifice of the infant's Eustachian tube, separating the adenoid from the tube-opening during the act of swallowing, thus causing a similar ventilation to take place as in the adult where the lumen of the tube, usually collapsed, opens during the act of swallowing because of the action of the pharyngeal muscles. In the child, were it not for this physiological growth, the tube would be open constantly.

Furthermore it is known that with the advance of years, as the temporal bone takes on more and more the adult type, the Eustachian tube becomes in course of growth longer, and more vertical in direction, its lumen becomes smaller, until its sides are in contact; and at this time—in the normal, healthy child—the adenoid remaining normal, then atrophies until it practically disappears; its physiological function as a block to the tube no longer being necessary.

With these things in mind, I hold the adenoid not of etiological factor in the ear disease of childhood unless it is pathologically involved; but believe it rather a protection afforded the ear to keep food particles, etc., from lodging in the tube. It is a familiar experience to have children, whose ears had at some previous time suppurated, give evidence of a succession of ear attacks after adenoid removal. In this connection it may not be amiss to pause in the ruthless removal of adenoids, until satisfied that this growth is really a pathological factor in the ear disease, or is in itself giving symptoms which demand its removal.

The recurrence of the adenoid, for I hold it to recur, many opinions to the contrary notwithstanding, is a fact which I have seen, and I believe with Goerke[25] that it will recur, the regeneration depending upon the continued activity of the identical factors inducing increased functional capacity and an increased production of lymphocytes. It is interesting in this connection to hear Barth[26] report that of his material, ageing about one year, only 29-30%, of 202 patients having adenoids, presented middle ear disease, and Kerley,[27] reporting from his private practice 51 cases of otitis media suppurativa acuta, found adenoid vegetation present in only 6 cases.*

*Regarding frequency of adenoids among the school population of New York, Chappel found their existence in 3 per cent. of 2000 children; other observers — as reported by Lartigne and Nicoll (28) — find them among children as follows: Rosenberg, 9 per cent.; Wroblenski, 7 per cent.; Schmiegelow, 5 per cent.

This must not be construed as a protest against the removal of adenoid vegetation from the naso-pharynx where such is hypertrophied, and presents itself as an impediment to nasal respiration. A little experience in the examination of the pharyngeal cavities of normal children who have well-developed nasal bridges and whose appearance show none of the stigma of the mouth-breather, will serve to teach the amount of adenoid in the normal child.

Furthermore, referring to what I said under general diseases, especially tuberculosis and syphilis, the report of Brieger and Goerke[27] become more important. They studied the difference

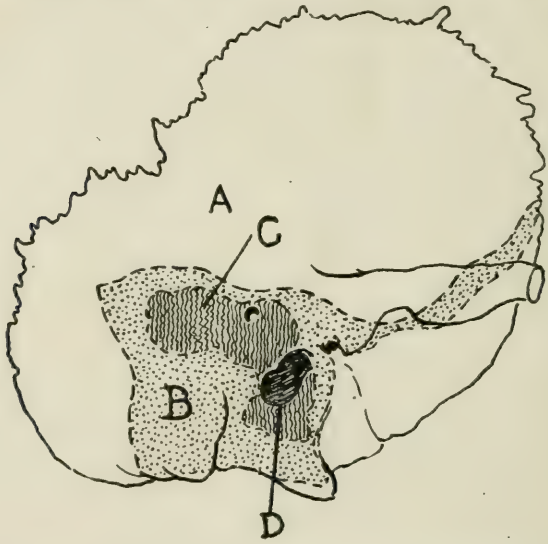


Fig. 3. Outline of the temporal bone, with shaded portion showing as cut in Fig. 7, on the external surface of the skull. A. Outline of the temporal bone. B. The line through which the section was made to expose. C. The middle ear and antrum on the external surface of the skull.

between the hypertrophied faucial tonsil and hereditary constitutional anomalies such as scrofula, tuberculosis and syphilis, and the so-called lymphatic constitution and infectious diseases. They point out that in a number of cases the clinical picture of tonsillar enlargement is due to tuberculosis, and that syphilitic processes occasionally simulate hyperplasia, and that the latter may occur as a side manifestation of hyperplastic processes in the lymphatics of the pharynx.

Furthermore, Lartigue and Nicoll[28] made examinations of 75 consecutive specimens of adenoids regarding tuberculosis and con-

cluded that tuberculosis of the adenoid is probably more frequent than supposed. They found 16% of their series containing the tubercle bacilli, and 10% giving characteristic lesions of tuberculosis.

Therefore I draw the conclusion that enlarged tonsils and adenoids and the ear suppurations are both due to the underlying constitutional condition, and that this condition, and not one of its manifestations (the adenoid) is the real factor of moment, and to expect to influence the ear in such cases by removing the adenoid (a manifestation of the constitutional condition) is bound to be unsuccessful.

On the other hand, when the adenoid is diseased, and every condition causing an inflammation of the mucous membrane of the pharynx—rhinitis, coryza, etc., the initial stages of the exanthemata --producing as they do first inflammation and then hyperplasia and hypertrophy, then the adenoid becomes a menace to the health of the ear. Nor do I think Mackenzie's statement an overestimate when he says that the discovery of the adenoid has probably saved nearly 100,000 individuals from deafness.

Regarding its presence in cases of disturbances of hearing, Abeles reports it as 57%, while Halbeis places it at 47% (cited by Zappert.[20])

Having discussed the anatomical peculiarities of the lymphatic tissue of the pharynx, the anatomical peculiarities of the juvenile ear presents factors which inherently are of etiological moment, not only as tending to account for the prevalency, but also as being more or less directly causative factors in the suppurative ear diseases of infancy and childhood. These factors may be briefly summarized as follows:

1. Characteristic features of the juvenile Eustachian tube.
2. Peculiar behavior of the petrosal pyramid during the developmental period.
3. Characteristics of the juvenile tympanic cavity and its contents.

Intelligently to understand these three factors, I briefly present the anatomy of the infant's and child's organ of hearing. This we find divided according to its physiology into a sound perceiving and a sound conducting portion. Thus, there is an external, middle and internal ear, the external and the middle relating to sound conduction, and the internal to sound perception.

To the external ear are counted the external auditory canals and concha; to the middle, the drum, ossicles, the tympanum and its correlated cavities leading into and including the mastoid cells on the one hand, and the opening into and including the Eustachian tube on the other. These all constitute the sound conducting apparatus.

To the internal ear, whose physiological function is sound perception and recognition, are counted the entire auditory nervous system, from its centre in the brain to its endings in the labyrinth, the cochlea, ampulla and semi-circular canals constituting the labyrinth.

The most characteristic feature of the Eustachian tube in the young is its shortness; according to Eitelberg,[30] its length is 19 mm., and Schengelidze[31] places it at 22 mm. in contradistinction to that of adults, which Politzer estimates at 34-36 mm. A further characteristic of the tube is its horizontal plane of direction, while in the adult it inclines away from the horizontal. Its lumen is wider, and the examinations of Schengelidze demonstrated that the lumen constantly grew smaller as the child aged. Its tympanic orifice is placed at a higher level than it is found to have in the adult; while at its pharyngeal end, the protuberance, so well marked in adult life, is never prominent in childhood. These characteristics of the tube have already been alluded to in the discussion of the adenoid in childhood.

Fabricinab Aquapendente,[32] and the older writers already were cognizant of the fact that different conditions prevailed in the tympanum of the infant and child than was to be found among adults. This refers more to the contents than to the bony wall of the cavity. They held that the tympanum was filled with mucous, or phlegm, or a reddish fluid. (Linke.[33])

Following the researches of v. Troltsch,[34] one of the first to point out the embryonal character of the tissue filling the tympanum, others, among whom Wendt,[35] Böke,[36] Weiss,[37] Kutscharianz,[38] and more recently Preysing[39] and Gomperz,[40] are noted, although differing as to histological details, in the main substantiated the fact that the contents of this cavity in early infancy are of an embryonal character.

Gomperz's conclusion, based upon 50 autopsies in older children, while establishing the existence of a cavity as such, still finds re-

mains of embryonal tissue, especially at the malleo-incudial articulation.

Without entering the discussion as to histological details, suffice it to say that this embryonal tissue, because of its retrogressive change, easily furnishes the soil for the propagation of invading micro-organisms, and thus the character of the tympanic contents in the young constitutes a factor in the etiology of the suppurations of the middle ear in childhood.

The upper wall of the tympanum (tegmen tympani) presents in the very young a fissure (the petro-squamous) situated between the petrosa and the squamous bones, extending backward as far as the mastoid antrum. It is traversed by a process of dura which

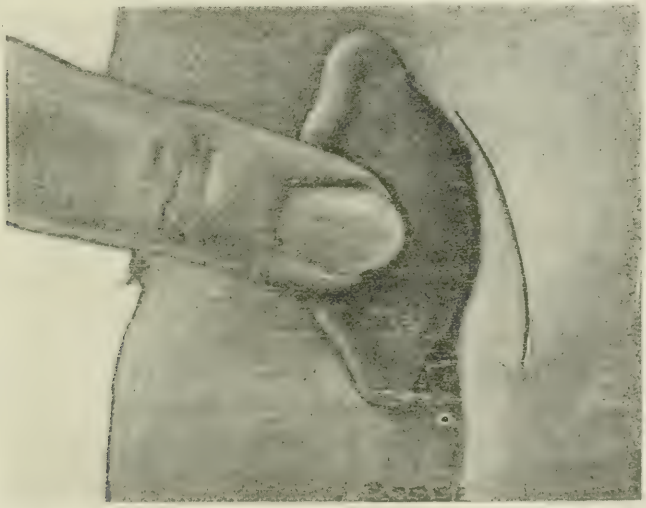


Fig. 4. Showing the line of primary incision from the middle of the mastoid tip upward. The auricle turned forward.

establishes communication between the lining of the tympanum and the cranial cavities; this dural process carries 5 branches of the middle meningeal artery for distribution along the upper wall of the tympanic cavity. This fissure closes at about the fifth month of ossification, until later in age the child presents only the remains of this fissure as the petro-squamosal suture. As a route by which infection may travel from the tympanum toward the cranial cavity, this fissure and it resultant suture become of importance.

There is another route for intracranial invasion in the young. This is the subarcuate hiatus, an orifice opening from the tem-

poral bone into the cranial cavity about 4 mm. in width, situated immediately under and to the side of the upper semi-circular canal, almost at the edge of the petrosal section of the bone. In adults, only a scanty remnant of this hiatus obtains.

The lower wall or floor of the tympanum is a thin bony partition covering the jugular bulb. In the young it is a thin, transparent plate. I have often found the mucous membrane of the tympanum in direct contact with the bulb, the intervening plate of bone either totally absent, or having distinct defects. (In 25 specimens examined in special regard to this point, I found the plate partially or wholly absent 4 times in 10 specimens ranging from birth to 3 years, and 5 times in all the specimens ranging in age from birth to 14 years.) Urbantschitsch[41] holds this as more or less of a common occurrence under 5 years of age, and Bryant[42] calls attention to the possibility of puncturing the jugular bulb during paracentesis through this thin bony partition; Dench[43] and Gerber[44] citing cases of such accidents.

The other walls of the tympanum present nothing characteristic of the young. The external wall (the drum) will be referred to below.

In size the tympanum does not materially differ from the adult. (Bryant.[42])

Regarding the rudimentary mastoid process, it is found at an early age, situated close behind and somewhat over the external auditory canal, gradually shifting its position to the rear of the external auditory meatus,, and is only fully developed about puberty.

The mastoid cells develop individually different characteristics in different children. Many claim absence of cells in the child's mastoid, but the total absence of pneumatic cells, together with the preponderance of diploetic cavernous spaces, is to be regarded as a sort of disturbance of development (Koerner.[45]) The newlyborn, acording to Koerner, present a fully developed antrum but no mastoid process proper, its existence is indicated by a protuberance. In this observer's opinion the mastoid cells' development progresses from the antrum, at first backward, later on toward the protuberance, until at the end of the first year pneumatic cells are apparent and begin to develop. At the third year the mastoid may already closely resemble the adult type, and I recently had occasion to operate upon a female child (2 years) whose mastoid presented no deviation at all from the adult type,

being not only extensive in area and fully developed, but having mastoid cells throughout.

In two of my dissections, one made on a specimen of 1½ years, the other at about 2 years, I was able to demonstrate an anterior and posterior grouping around a central plate of bone. I find that Barbarian[46] cites Schwartz and Eysell as describing such grouping and an intervening plate with a posterior inferior group of cells concealed below and behind the antrum. He states that this plate lies in the course of the petro-squamosal suture. I fail to substantiate this claim from the meagre material so far at hand.

The external wall of the tympanum is made up of the membrana tympani. This differs but slightly from the adult, regarding size, but its form is slightly more oval. Its position changes gradually, from birth, as the child ages. Generally speaking it is almost horizontal at birth, and takes upon itself a more vertical position as age advances.

Many observers hold that the infantile drum is of greater thickness and density than that of the adult, and to this anatomical fact these observers lay the fact that otitis medias in children run their course occasionally without producing perforation, but I believe that the consistency of the drum in the child is as variable as we find it among adults, and I have as often found it, delicate, thin, and translucent in the one class of patients as in the other. The ossicles present nothing characteristic of the young.

From the drum, the external auditory canal connects the internal parts with the external ear or concha.

This canal, at birth only a ring of bone, continued to the concha by soft parts, the ring of bone develops, becoming a broader segment as age increases, until between the third and the sixth year, it takes on adult characteristics.

Oftener than I supposed (I found it 4 times in 10 adults' bones and almost ¼ of all childrens' bones examined) there remain defects in the ossification of the external auditory canal, and these are of importance, because occasionally they are a point at which pus will break through from suppuration of glandular tissue in this region, which, exuding at the meatus, simulates a purulent otorrhoea. [I had such a case under treatment last winter, where the child (male 4 years) was sent to me for treatment of a persistent purulent otorrhoea, which had followed an attack of mumps. The glands in the neighboring regions were still swollen, and

the external auditory canal, filled with pus, its walls excoriated. Only after careful cleansing and inspection was the fistulous opening in the lower anterior portion of the external auditory canal noted, about 1-3 of its length inward. When a small probe entered this opening, and further inspection demonstrated a normal drum, the diagnosis was clear. A counter opening over the swollen gland, followed by appropriate treatment cured the case.]

Of the external auditory canal it may be stated that its walls are almost in apposition in the very young, and that the lumen widens as the child ages. Its roof boundary is the external surface of the skull (superficies meatus.)

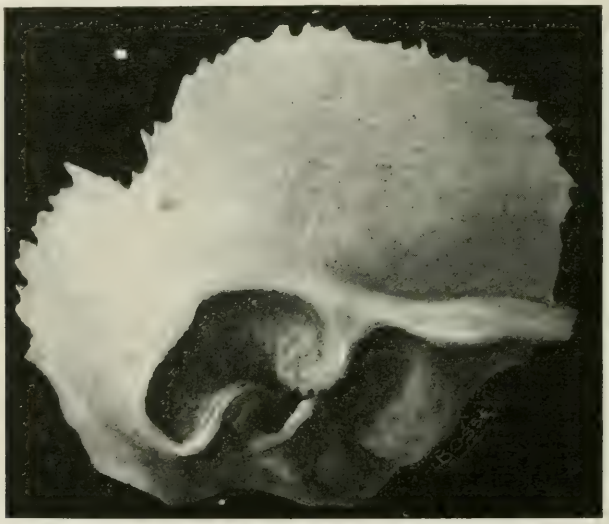


Fig. 5. The bone surgery completed in the performance of the simple tympanomastoid operation on the two-and-a-half-year-old subject. (Drawing from author's specimen.)

Symington,[47] in an exhaustive study of the meatus of childhood gives the length of the canal for the various ages as follows:

Age.	Floor.	Roof.
2 mos.	17 mm.	13 mm.
6 mos.	19 mm.	14 mm.
1 yr.	20 mm.	15 mm.
2 yrs.	22 mm.	16 mm.
5 yrs.	23 mm.	17 mm.
6 yrs.	24 mm.	17 mm.

Studying this table, the angle of inclination of the drum to the horizontal axis of the canal is outlined.

Finally, it is to be noted of the squama, as a characteristic of the young, that it is bent upon itself, forming an agle. This not only becomes of importance in the surgery of this region in the young, for this bent plate overlies the aditus, but from the direction of this superficies meatus and the direction which it gives to the external auditory canal it causes the concha-attachment to the underlying bony parts to lie at a relatively higher level than the level of the tympanic membrane, a fact to which I recently called attention,[48] and which is in contradistinction to the condition obtaining in the adult.

The concha, while not fully developed as regards size, is relatively typical of the adult in other particulars, and the labyrinth, fully grown and developed at birth, both as regards form and size, presents no special characteristic of early age.

Before closing this section, I must refer to the defects noted by many observers (Koerner and others) in the bony canal incasing the facial. These defects lay the contained nerve open to attacks by the purulent contents of a diseased temporal bone, with resultant early paralytic phenomena. This point of emergence of the nerve from the skull (stylo-mastoid foramen) is very superficial, to which fact I will revert later when discussing surgery.

Finally summing up the factors which make the anatomical peculiarities of the infants' and childs' temporal bone, etiologically important, and as such one of the causes for the prevalency of suppurative middle ear disease we find.

1. The anatomical characteristics of the petrosa with its contained auditory apparatus behaves while in the developmental period differently than it does in adults. (Baginsky.[49])

2. The characteristics of the tympanum, its freer communication with the nasopharynx, and cranial cavity, together with the retrogressive changes in its lining membrane and contents.

3. The frequency of dehiscencies and abnormalities. These constitute the anatomical factors underlying the frequency of middle ear suppuration in childhood.

III. PATHOLOGY.

It is impossible when speaking of the factors producing prevalency of ear suppuration in childhood, not to bring out the pathological factors, for these two are interwoven, and while the factors already discussed may be regarded as remote causes for

the disease, the immediate cause is to be found in the invading pathogenic organisms.

The development of the pathological study of suppurative conditions of the middle ear of childhood is largely the work of Zaufal,[50], Netter,[51] Weichselbaum,[52] Gradenigo,[53] and Penzo,[53] Brieger[54] and Prysing.[56]

Many micro-organisms are to be found in causal relationship to the condition; the streptococcus pyogeneus; the pneumococcus, the staphylococcus pyogeneus, the pseudo-influenza bacillus (Kossel[56]), Fraenkel's pneumo-bacillus, Friedlander's bacilli; the bacillus pyocyaneus, the tubercle bacillus (Habermann[57]), the gonococcus (Flesch[58]), the Vincent's bacillus and spirillum (Held[59]), are among those met with.

Gomperz[40] is authority for the statement that the virulence of many bacteria is reduced in the tympanic cavity. Regarding the order of frequency with which particular organisms are found in the middle ear, a divergence of opinion exists. Weiss[60] found the diplococcus pneumonia in 42% of cases; the streptococcus pyogenes in 39%; the staphylococcus albus in 27%; the staphylococcus aureus in 12% of his cases; on the other hand, Preysing studying 121 infected ears found the pneumococcus infection ranging as high as 92%; reading Preysing's results with the observations of Schcniglidse[61]—who making 90 autopsies found pneumonia accompanied by otitis in 91.6% of the cases; and we are of the opinion that the pneumoccoccus is the most frequent invading organism in the middle ear of the child. Furthermore, regarding the invading organism, I recall the old theory of Zaufal, that primary otitis suppurations are always from a pneumococcus infection, while secondary involvements of the ear almost invariably belong to the streptococcus infections.

The relative rarety of the staphylococcus is to be ascribed to the fact that the tympanic infection usually is an acute process, and in acute processes one rarely finds staphylococci.

In passing, let me note that the coincidence of otitis and pneumonias was commented on as early as 1864 (Streckheisen[62]) and 1868 (Wreden) and recently Gomperz in 1906 holds the common cause of both pneumonia and otitis to be the grippe infection.

Having thus briefly sketched the offending organisms we will next consider the route of invasion.

In the child this is usually through the Eustachian tube. This tube, the open door to the middle ear, its protective arrangements paralyzed, the disease and its causative agent travels by contiguity to the tympanum, and there, by reason of the character of the tissues which undergoing retroactive changes furnish a ready soil for germ propogation,—the micro-organism take hold, and produce their malignant effects, whose results are classified as purulent otitis medias.

IV. Clinical Picture.

The clinical picture of the onset of a typical case of middle ear suppuration is usually sudden, with or without demonstrable cause, or coincidental with general systemic disturbances. There

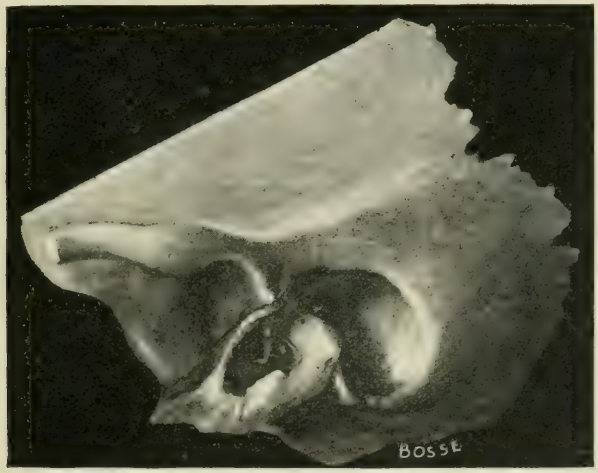


Fig. 6. The radical operation (bone) completed up to the removal of the ossicles. The point of breaking through from the antral opening to the tympanic cavity is shown. The anterior and inferior wall of the osseous external auditory canal has been removed to better expose the unremoved ossicles. (Drawing from author's specimen, aged about two and a half years.)

may be headache, languor or lassitude, and more or less pain on the affected side. Very often both ears are affected simultaneously although to varying degrees. Fever, and in children old enough to have it determined, an interference with hearing is obtained, proportionate to the degree of inflammation present. Rigidity of the neck is sometimes noted. This is the clinical picture in older children.

But in the young infant and child, where headache cannot be expressed, and where pain remains unlocalized, the question of the clinical picture becomes more difficult.

Generally speaking, every child exhibiting uneasiness, fever, symptoms of intracranial irritation, should be examined as to its ears, unless some distinct pathological condition is present to account for the symptoms.

Even the early writers among them Schwartz[63] (1825), Meissner[64] (1828), Helfft[65] (1847) and Hauner[66] (1863), without the aid of otoscopic examinations, pointed out the frequency with which childrens' ears were affected and the ease with which such conditions might be overlooked.

Vomiting, somnolence, uneasiness, delirium and coma were the grave symptoms which the ear disease brought in its train, these all subsiding with the advent of the otorrhoea. Even v. Troeltsch, believing an otoscopic examination almost impossible, made his diagnosis per exclusion. His construction of the clinical picture, as cited by Gomperz, are worthy of repetition.

Uneasiness, restless sleep interrupted at intervals by wakeful periods, the child rolling its head about on the pillow or boring into it, with an occasional characteristic cry of pain, this cry repeating itself at the least jostling or shaking—these constitute a clinical picture whose recognition is easy to the observer of today.

To touch the affected ear produces pain-cries. There are frequent remissions, the pauses lasting from hours to days. The loss of appetite is however constant. The act of swallowing may cause pain (v. Troltsch, Gomperz). I have observed a nursing infant attempt to suck and after repeated trials let the nipple go and start to cry. Gomperz saw the same thing, and further reports that when fed with a spoon the child under his observation took the nourishment.

In very young infants, gastro-intestinal disturbances, manifest themselves, due to the absorption of toxins from the tympanic cavity, and the stools soon increase in frequency accompanied or not with vomiting. Following the gastro-intestinal derangements, there is less of weight (Hartmann[67]), (Schengelidze[68]).

Qoppert[69] held the vomiting to be due to hyperaemia of the dura and partly due to irritation of the labyrinth, and Gomperz (l. c.) holds this symptom as the first signal requiring an answer in the form of a paracentesis.

If the condition remains unrelieved, the fever continues high (if patient is more than one month old) and finally after the devel-

opment of antrum empyema, or mastoiditis, cerebral symptoms come on, followed by coma and finally death.

It must be remembered that bowels and lungs may be involved either coincidentally, or prior to, or as a resultant involvement from the ear disease, and in any of such cases the difficulty is multiplied when attempting to analyze the clinical picture and differentiate the general, from the local disease.

To take up a mooted point. Regarding intestinal disturbances, it is a fact that many observers have seen these, coincidental to ear disease (also personal observations); but, while same have held the bowel derangements and vomiting as causative of the ear trouble (Goppert[70]), others held that the bowel derangements were the result of the ear trouble, (Preysing, Ponfick,[71] Hartmann[72]), and Jacobi [73] believes both views have foundations on fact.

Preysing's conclusions seem to me the most trustworthy. He holds the diarrhoea to be a kind of septic diarrhoea and recalls to our minds the fact that the pneumoccoccus—which is the most frequently offending micro-organism—belong to that class of streptococci which are the causal agents of sepsis, it having been demonstrated that streptococci toxins cause diarrhoea and also ecchymosis of serous membranes, (Homer and Laitinen[74].)

Whatever may be the cause of the gastro-intestinal disturbances, it remains a fact that one often sees the onset and determination of the disease of the ear, with the onset and termination of bowel derangement, and I hold with Gomperz that any one who has observed this will be impressed with the fact that a direct effect is produced by ear disease upon the entire body, the bowel troubles being a manifestation of this systemic effect.

It is noteworthy in this connection that, of the material reported, a large majority of the children found to be suffering from middle ear suppurations at autopsy were undeveloped, marasmic, atrophic and under nourished.

I have observed cases where the nourishment of the children suffered, and where there seemed to be general malnutrition, anaemia, and generally a feeling of non-well being, in cases wherein the attending physician could not locate the disturbing factor, and the otoscopic examination demonstrated only a thickened drum; these cases remained obscure until an examination of the pharynx evidenced a purulent exudate coming from the Eustachian tube. (One case, girl 3 years of age, and two male children, aged 2 and 3

years respectively, in one of which—the younger—purulent exudate also came from the nostril on the affected side).

Finally the advent of otorrhoea. In younger children otorrhoea does not present itself early. This has been held to be due to greater thickness of the child's drum. (Jacobi and others). I do not believe that the thickness of the drum plays any important role. But the wide open tube, affording partial drainage toward the pharynx saves the drum from early perforation, and thus the ear disease may be present for some time, the deleterious products of the suppuration absorbed and swallowed to the detriment of the child's health before the presence of otorrhoea points the way to the diagnosis by the attending physician.

To summarize:—

When a child, without demonstrable cause, while under usual diet, begins to run fever, is restless, rolls its head, and refuses to

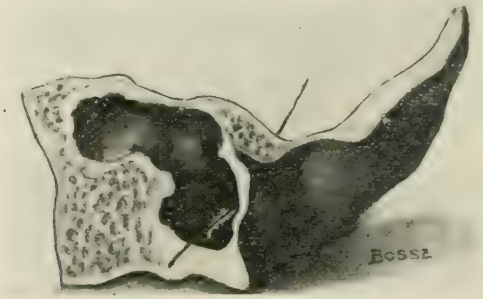


Fig. 7. A. Section cut as indicated in the shaded portion of Fig. 3, showing the middle ear (tympanic cavity), aditus ad antrum and antrum. A bristle has been passed through the existing defect in the tympanic floor, coming out through the roof of the jugular bulb. (Author's specimen, aged two years.)

drink, meanwhile giving evidence of bowel derangement with consequent loss of weight, unless the clinical picture points in a definite pathological direction—then the ears should be subjected to rigid examination to determine their condition. Among older children, the localization of pain and persistence of fever, with negative results from general examination, points to ear involvement.

Finally, because a pathologic process in the ears of young children may be present for a longer period than the obtainable history would suggest, it results that many cases already have the elements of chronicity when discovered, bone necrosis already at this time having begun.

V. THE COURSE.

The course of middle ear suppuration in childhood are dependent upon the causal facts influenced by the patient's age and inherent powers of resistence. The latter a result in a great measure of inherited tendencies.

The duration of suppuration lasts from days to weeks. I have seen purulent cases, with bacteria demonstrated, entirely dry, perforation closed within a week from onset. Regarding duration we can safely say that the earlier the child receives rational treatment the earlier the cure, excepting such cases where constitutional taint of tuberculosis or syphilis play a role.

It must be noted, however, that many cases linger along in spite of treatment, and in many of these, there is usually to be found enlarged faucial tonsils, in others, the children belong to the undernourished type, or they present the above mentioned constitutional stigma.

VI. THE OTOSCOPIC EXAMINATION.

I have endeavored to so fully draw the clincial picture that there remains but to call attention to the otoscopic examination, and the question of diagnosis is settled.

I believe the otoscopic examination if carefully performed will always give valuable additional data, and hold that in 80% of all children, and 100% of all children above one years' age, an otoscopic picture is obtainable.

To aid in procuring this, I have had constructed a speculem (Fig. 9-10) shaped like a truncated cone, its sides concave, whose smaller end is finished off at an angle of 45%. This allows of a wider field of vision for the same thickness of speculem, than would be obtainable were this end cut straight.

For illumination. we have an incandescent light attached, so that children can be examined in bed, or in arms, without reliance on reflected light. And to any one who has attempted to examine a struggling infant, this little instrument will appeal as a distinct advantage.*

The drum pictures differ in one way from the findings in adults except that all pictures are seen fore-shortened because of the great incline toward the horizontal which the child's drum presents.

Instrument made by the Wappler Electric Controller Company of New York City.

VII. COMPLICATIONS.

Besides the complications, general in nature, which the middle ear suppuration in childhood presents, we have periosteal abscesses, mastoid antrum empyemas, and mastoid process involvements, brain abscesses, suppurations of the labyrinth, and sinus phlebites.

To discuss these in detail is beyond the scope of this work, but I will briefly note some characteristics peculiar to childhood.

Periosteal abscess develops rather easily in childhood, because of the exceeding thinness of the external wall of the antrum. Gomperz finds the swellings presenting themselves more often above than behind the ear, in contradistinction to the position assumed in adults.

Empyemas of the mastoid process very often give few if any distinctive symptoms in early life and are easily overlooked, while in the acute empyemas of the mastoid antrum, they characteristically present (not always however) a rather profuse otorrhoea with a slight sanguineous tinge. The discharge is demonstrably coming away under pressure. Occasionally the soft canal wall is separated from its bony parts, by the extraversation of pus; and in one case seen by the writer, the drum remaining intact, the drum and soft canal became everted like the turned-in end of a glove finger, this presenting at the external auditory meatus, like a new growth.

Following free incision and removal of pus, with replacement of everted part of canal, healing resulted.

Swelling of the gland at the angle of the jaw, in a case presenting a particularly fetid otorrhoea, is occasionally the only symptom pointing to mastoid involvement in those with constitutional stigma, or in ill nourished marasmic children.

The external auditory canal usually narrows because of inflammatory swelling of its walls, presenting superficial ulceration from excoriations by the pus. I place more reliance on this narrowing of the canal, than the sinking of the posterior superior quadrant, as the former, in my experience, is the better guide in childhood.

Facial paralysis, coming on early, while it may presage great involvement of the mastoid, is not reliable, for the frequently occuring defects in its bony casing, easily lays the exposed nerve open to attack.

Brain abscess, presents nothing characteristic of childhood. Meningitis may develop through invasion of the Cavum Cranii through

perforation of the tegmen tympani; through the labyrinth and porus acusticus internus; or in younger children, through the various open sutures; or subarcuate hiatus. In the great majority of cases the meningitis is basilar, but it is tubercular when following a tubercular otitis.

Sinus involvement, presents no special characteristics, only the initial chill is often absent, replaced however by vomiting and convulsions, furthermore the fever is usually atypical.

VIII. TREATMENT.

Before discussing the treatment of suppurations of the middle ear in childhood, I will briefly dwell upon some prophylactic measures of moment, especially during the exanthemata.

The care of the ears during the attack of any of the exanthemata should be greatest during the period of greatest emaciation—

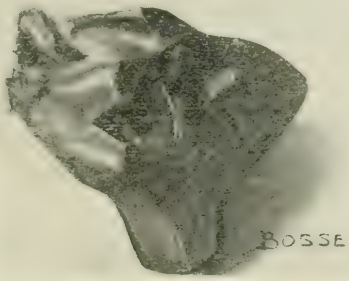


Fig. 8. A section of the temporal bone of a two-year-old subject, through the facial in its descending line. The distance of the facial from the drum is evident, and the protection to the nerve afforded by the horizontal semicircular canal is shown by the overhang of this canal. The relations of the ossicles as exposed to the semicircular canal is also shown. (Drawn from the author's specimen.)

usually when the disease has about run its course. Simultaneously with the time when kidney and other lesions are watched for, the ears should be guarded, and danger to them only considered as past when the entire rhino-pharynx has become normal in state and appearance.

Every effort should be directed to remove mucous or other exudate from the pharynx, through thorough cleansing with warmed alkaline washes, and in younger children by the use of swabs.

Swollen turbinates should be kept within bounds by the judicious use of adrenalin and minute doses of cocain locally applied.

The removal of hypertrophied adenoid tissue during the course of a general disease, is a question which must be decided individ-

nally for the given case. Among those advocating its removal at once we find Knyvett[75], and Schiebe[76] points out that the otorrhoea following exanthemata persists longer in resisting treatment, when adenoids and hypertrophied faucial tonsils are present than would otherwise obtain. Personally I have removed adenoids toward the end of an attack of scarlet fever in a case where restlessness, recurrence of fever, and reddening of the drum warned the attending physician that the ear was endangered. No otorrhoea resulted in this case. I may say however, that the child did not seem very much prostrated by its scarlatinal attack, and the relief afforded to nasal breathing, and the blood letting, consequent to the operation, may have materially helped the abortion of the threatened ear involvement.

The treatment of middle ear suppurations in infancy and childhood will not materially differ from that administered to adults, except as modified by the peculiarities of the parts in childhood.

Internal medication must of necessity, because of tender age, be cautious.

The application of leeches is condemned, but the application of tincture of iodine, painted on the skin behind the ear in the beginning stages, has brought the writer good results. The application of heat and cold are hardly practical in the younger patients. The pharynx must be kept clean; and pharyngeal and nasal obstruction must be minimized or entirely removed. (Ostman,[77] Weiss,[78] Love[79], and others.)

While formerly some differences of opinion existed as to the advisability of early paracentesis, I believe the profession is now as one in regarding it as the first measure to be undertaken. Nor is it necessary to wait until a purulent exudate is demonstrable before proceeding to paracentesis, it being my opinion that it is better to open the drum too early than too late, since when properly performed, under the rules of asepsis paracentesis presents no dangers.

The incision should be made in the inferior posterior quadrant, after thoroughly cleaning the external auditory canal.

In advanced cases, I employ a small suction apparatus which, under full illumination, draws the secretion (very often thick and fibrinous) from the middle ear into the canal from whence it is washed away. A wick of gauze is then introduced (private practice) into the incision, this gauze moistened with a weak bichloride

of mercury solution (1.5000) and the canal lightly tamponed, and over the whole a wet dressing and bandage applied. After 10 hours, systematic douches of (1.5000) bichloride of mercury given warm are administered.

If the secretions are thick and viscid, the douche is preceded by a few drops of diluted peroxide of hydrogen solution.

I desire to deprecate the insufflation of all powders, or the employment of evaporating solutions containing ether or alcoholic (solution of powders) as these leave a coating of their contents, after the volatilization of the liquid, on the canal and drum, and after repeated use, tend to block up the canal and prevent drainage.

When symptoms indicate the involvement of the antrum mastoidum (in infants) or the mastoid process in older children, provided the case is seen early in its course (within the first week) then I employ induced hyperaemia according to the method of Bier, and although this method has not yet been adopted universally, it early appealed to me as best suited in children's cases, and I feel that by its employment, I have seen cases clear up which otherwise would have come to operation[80].

In cases coming under observation later in their development, where fever persists, where pus is evidenced under high pressure, where granulations present early through the perforation, or grave symptoms of intracranial involvement supervene, I believe more is to be gained in the child's interest by early operation than the indefinite extension of the expectant treatment, and upon such cases I accordingly operate.

Whether to perform a simple or a radical operation must be determined for each case. With evident bone necrosis in childhood, the radical operation affords the better chance for recovery, for it must again be emphasized that underlying such, are usually a tubercular, scrofulous, or syphilitic basis, and the little patient is better off when these diseased areas are radically eviscerated.

Finally, it may be remarked as almost axiomatic of the treatment of suppurations of the middle ear in infancy and childhood, that the efficiency of treatment decreases progressively with the duration of the ear disease.

IX. TYMPANO-MASTOID SURGERY.

It is not my purpose here to rewrite the anatomy of the temporal bone, but to call attention to certain peculiarities of the child's tem-

poro-mastoid region in sufficiently detailed a manner to make clear why I have adopted the procedures detailed below.

As already explained in the chapter on anatomy, the external auditory canal and its relations to the surrounding bony structures are of vital importance to the surgery of this region. Closely adherent to the superficies meatus, its roof follows the contour of the squamosa. Its direction from the concha is, generally speaking, downward, forward and inward. See Figure 1.

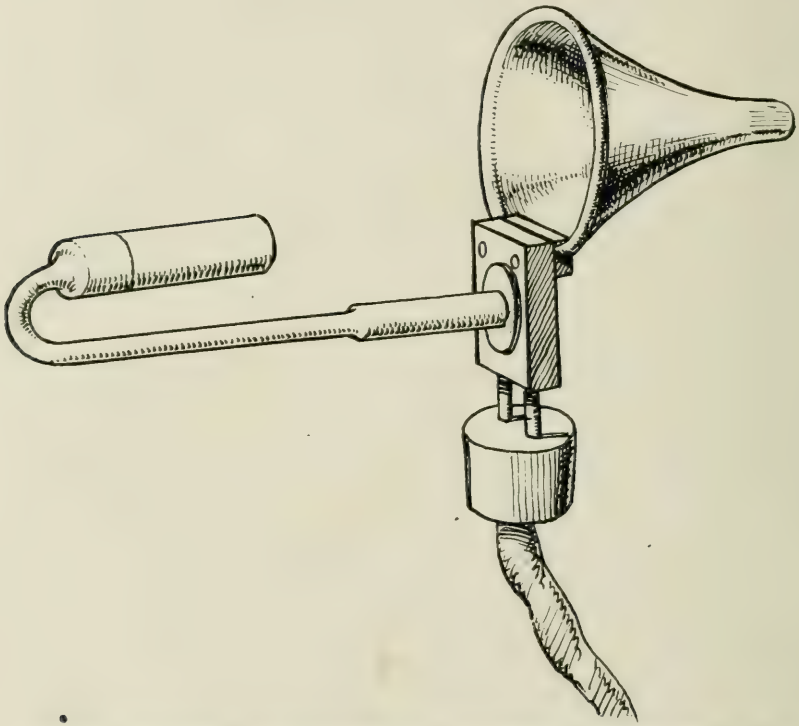


Fig. 9. Drawing of the author's speculum for examining children's ears, with the Wappler electric attachment for direct illumination.

Noteworthy is the fact that in early age the roof of the canal is bounded superficially by the superficies meatus. This superficies meatus is the bent part of the squamous plate overlying the aditus. See Figure 2.

From the direction of the superficies meatus and the external auditory canal, it follows that the attachment of the auricle to the underlying bony parts is at a higher level than that of the tympanic membrane, and the line of incision which is to give approach to

the middle ear in the child will have to be brought to a higher level than the annulus tympanicus, and the superior wall of the external auditory must be separated at its roof from the superficies meatus.

The separation of the soft external auditory canal from the superficies meatus must be carefully carried out in retracting the soft parts to expose the bony surface above the annulus tympanicus. I have never found it necessary to incise the canal to view the tympanic membrane through it, so as to identify it, as recommended by S. Oppenheimer,[1] and I consider the possibility of infecting the cartilaginous and soft parts of the external ear as a danger, when such a procedure is attempted.

The next anatomical structure peculiar to the young, the existence of which, so far as this stage of the operative procedure is concerned, will be of importance only when operating on infants and young children, is the masto-squamous suture. This suture is found externally on the temporal bone, in the very young unossified, in older subjects ossified, but equally well marked, extending downward from a small distance above the temporal ridge, marking off the squamous plate from the more or less rudimentary mastoid process. It occasionally persists through life, is usually wide in infancy and early childhood, and the primary incision, when made down through the skin to the bone, should be carried backward toward the mastoid tip to prevent the knife from entering the squamo-mastoid nerve at its point of emergence from the skull, this nerve having a very superficial point of emergence during the early period of life. Oppenheimer (l. c.) quotes Stiles as having called attention to this danger, and McEwen found this suture persistent on the operation table in cases aged twelve, thirteen, fourteen, eighteen and twenty-nine years.

On the inner aspect of the temporal bone, along the line of the squamo mastoid suture, we find the petro-squamosal suture, containing, in the young, the petro-squamosal sinus. While this is not of very great surgical importance, still, when retracting the soft parts, should the petro-squamosal sinus persist, its injury may result in directly infecting the cranial cavity with its contents. Furthermore, the squamo-mastoid suture marks the lower and posterior limit of the descending plate of the squama, and this is the line behind which we must expect to find the sigmoid sinus. No matter how small the temporal bone, it is rare, indeed, to find the sinus

further foreward than the external evidence presented by this suture. See Figure 2.

Our next step in the procedure is to determine the place for opening the antrum. In very young children the antrum is all there is of the mastoid.

The mastoid antrum and middle ear are much nearer the surface of the skull in the young than in the adult, and their position relative to the overlying parts differs so much from the adult type that other landmarks must be relied upon to locate it. Neither the spine of Henle nor the suprameatal triangle is a sufficiently constant guide for the safe performance of the operation. Of all the landmarks, the most constant is the ridge of bone formed by the posterior root of the zygoma. This ridge, by its lower border, indicates the level of the roof of the mastoid antrum, above which, a few lines in distance, will be found the level of the base of the brain. This ridge, sometimes designated as the supra-mastoidal crest, is formed by the obliquity of the angle between the auditory plate of the squama (in the young), and the rest of the squama. It is sufficiently constant to furnish a reliable guide to the upper limit of safety in entering the antrum and avoiding the cranial cavity. See Figure 2.

The tympanic cavity communicates with the antrum by means of the aditus ad antrum and the epitympanic space, commonly and erroneously designated the attic. The epitympanic space is that portion of the tympanic cavity lying superior to a line passing through the short process of the malleus, and containing the head and neck of this ossicle and the short process and body of the incus. Locating the epitympanic space on the external surface of the skull, we find it, in the child, directly above the tympanic membrane, almost in the middle of the upper part of the arch spanning the annulus tympanicus. Posterior to it, the antrum inclines slightly toward the back of the head as the child ages, until this cavity presents itself as usually found in the adult type of the bone. See Figures 3 and 7.

The Simple Operation:

Therefore, the soft parts retracted, the external auditory canal separated from the underlying bone, the root of the zygoma and the supra-mastoidal crest identified, and the position of the squamo-mastoidal suture determined, we must proceed to demolish the outer wall of the antrum, which lying high, is opened at a point a

few lines behind and above a plane drawn through the handle of the malleus. Opening the antrum thus, the facial nerve is avoided, being protected in this region by the overhanging horizontal semicircular canal. (See Figure 8.) From this opening the cortex is easily removed backward toward the squamo-mastoidal suture, the underlying bone and cell detritus removed with the spoon curette and sponges, and the purulent contents completely eviscerated. As we reach the squamo-mastoidal suture, a hard plate of bone is encountered—the inner table of the skull—and under this the sinus is to be expected. I have never found it necessary to curette the aditus in the simple operation on the child, nor do I permit the

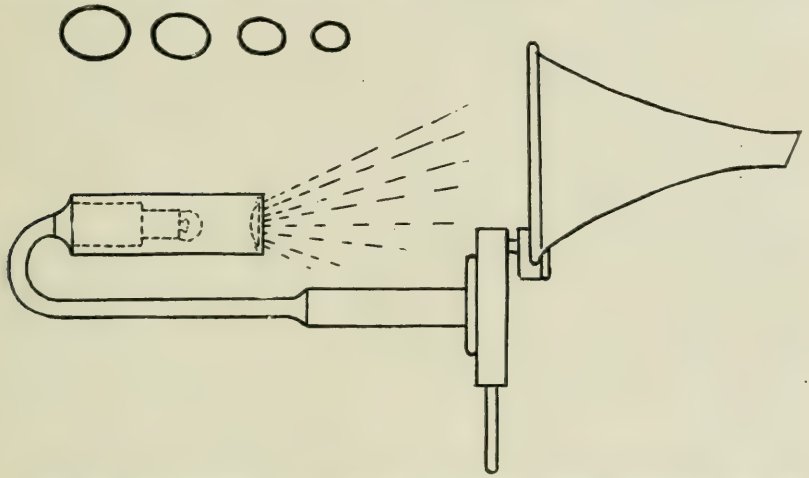


Fig. 10. Same as Fig. 9, only drawn in outline to explain mechanical parts and give outlines of the areas to be seen with the various sizes of specula.

curette to enter the epitympanic space. The danger of dislocating the ossicles, or of injuring the facial nerve has always seemed to me too great to hazard such risks.

The operation as above described affords sufficient counter-opening to effect drainage, paracentesis having been performed before the case comes to the operating table.

The usual result from this operative procedure is to give clean granulating surfaces which, under proper precautions during the post-operative period, the skin wound being kept open until the granulations reach its level, heals entirely without trouble or incident. See Figure 5.

To summarize the successive steps of the simple tympano-mastoid operation on the young:

1. Primary incision through the skin and soft parts, down through the periosteum to the bone, the incision starting directly over the middle of the mastoid tip and being carried by a curvilinear sweep toward the auricular attachment upward to the upper pole of its insertion, distant one-quarter inch from it. (See Figure 4.) I prefer to make this incision from below upward, for this furnishes another guard against injury to the facial nerve at its point of emergence from the skull.

2. Gentle efforts to retract the wound edges are now begun. Periosteum is pushed backward beyond the squamo-mastoid suture; the auricle gently bent forward on itself, is pulled forward and downward; the soft external auditory canal is carefully separated at its roof from its adhesions to the superficies meatus; and then the external ear, with its canal, is pushed forward and downward until the tympanic membrane is seen. This must positively be identified. Bleeding points have in the meanwhile been secured, and the operative field is now exposed.

3. We next identify the landmarks, the zygomatic root, the ridge running from it, and the squamo-mastoid suture. The point of selection for entering the mastoid antrum is then located, provided no fistulous opening already points the way to the suppurating cavity. This point of selection is directly above and a little behind the arch of bone spanning the upper pole of the membrana tympani, and with a slight tap of the chisel the bony wall is broken through. Occasionally, a "spongy spot" is discernible, and many authors follow this as a guide through which to enter the antrum. Personally, I have not found it a reliable sign. Many children's bones were found to be of so vascular a structure that its demarcation from the surrounding bone tissue was not always clear.

4. The antrum open, the cortex is easily removed, by means of a curette, backward to the tip, care being exercised as the squamo-mastoid suture is approached. The tip is removed, leaving only a shell of bone, and the operation is completed by removing all diseased tissue, not forgetting the cells at the root of the zygoma. Unless these cells are examined, and, if diseased, eviscerated, the object for which the operation was undertaken will be defeated.

5. The operative procedure closes by lightly packing the bone cavity with gauze. One strip of iodoform gauze is placed in the deep part, and covered with plain sterile gauze in one strip, the end of which should protrude beyond the skin wound and be lost

in the external dressings. This single strip packing will greatly facilitate proper drainage, and it is a better procedure than packing the cavity with small pieces. A strip of plain gauze is placed in the external auditory canal, and the external dressings are applied.

Regarding the change of dressings, it can be said that the longer the primary dressings are left in place the better, and although the outside dressing and bandage may require change because of soiling, it is better to wait until the mastoid cavity has begun to discharge (after five days), and the packing becomes thoroughly wet, before its removal is attempted, because then the change of dressing is less painful, and consequently will not be resented by the young patient. However, should there be any indication for earlier inspection of the wound cavity, this should be undertaken as soon as it is required.

The Radical Operation:

The location of the landmarks, and the initial steps leading the way to the antrum, are similar when performing the radical operation to those in the simple procedure as above described. The peculiarities of the temporal bone of the young which it is necessary to mark for the correct and safe performance of the radical operation, are: first, the spot where the least danger exists in connecting the antral cavity, as opened in the simple procedure, with the tympanic cavity, so as to form one connecting cavity. Here the danger of dislocating the ossicles plays no role, but the chance of injury to the facial nerve is a potent danger. Every effort should be made to avoid it, since, as McEwen[*] has pointed out, injury to the facial nerve in infancy or during the active developmental period of the facial bones and muscles, will arrest growth on that side and lead to disfigurements in excess of that caused only by motorial paralysis.

The nearer we make the connecting passage to the zygomatic root the safer the procedure. I have repeatedly tried this on the cadaver and have, since its verification, used it at the operating table, and I now feel that by breaking through from the antral opening directly above or near the position held by the malleus there is afforded an absolutely safe route for the primary step; the facial nerve, at this point having passed into the deeper parts of the petrosal pyramid, is not within range of the chisel of gouge, when these are carefully used.

We break down the root of the zygoma, and remove the remaining portions of the ossicular chain, endeavoring to keep the stapes intact and in situ, or at least to maintain its foot-plate in position. See Figure 6.

The antral opening connected with the tympanic cavity, this cavity itself next receives our attention. In the child this cavity presents two characteristics peculiar to its stage of development. First, the mouth of the Eustachian tube is wide and easily entered by a small curette; the bony walls are exceedingly thin, and great care must be exercised lest in curetting the bony wall separating it from the cartoid, this wall be injured to the damage of that vessel.

The second characteristic of the child's tympanic cavity is its floor which, covering the jugular bulb, is a very thin plate of bone. I have often found defects in this floor, with the dome of the jugular bulb in direct contact with the lining membrane of the tympanum, hence I recommend the utmost care when curetting in this neighborhood. See Figure 7.

Unhappily, the amount of material is not large enough (see anatomy section), to warrant me in citing my statistics, but the presence of these dehiscences in the floor of the tympanic cavity has occured sufficiently often to impress upon me the danger of rupturing the jugular bulb, and I have discarded all sharp instruments in this region, contending myself with a thorough sponging with gauze until satisfied that the bony floor is intact.

The tympanic cavity cleared of granulations and detritus, the partition dividing it from the antrum is removed under full illumination, and the facial canal, standing white and hard (at times evidencing dehiscences in its wall with the nerve exposed), is located against the darker spongy bone. The little bridge between antral opening and tympanic cavity is gradually broken away in small pieces and the radical operation—that is, the formation of a large, irregular cavity, merging tympanic, antral and mastoid cavities—is finished.

That is to say, the bone surgery is now completed, but to insure the best results a flap is cut from the soft tissues of the external auditory canal, so that the cavity may more rapidly become filled by granulations and its interior epidermatized. This flap may be so constructed as to provide easy access for treating any part of the bony cavity during the post-operative period.

The flap best adapted to children's cases has proved, in my experience, to be that devised by Panse. After the flap has been cut the edges are turned back and held in place by small pledgets of gauze. I find it best, in children, not to suture the post-auricular wound, but to encourage healing by granulation. The wound is packed with regard to holding the flaps in position, in a way similar to that described for the packing of the simple operation. The dressings remain unchanged until after the sixth day, when I have usually found the flap edges adherent. Further treatment of the wound differs in no way from that after the simple procedure.

The advantage of this method of handling the flap lies in the fact that it is cut at the time of operation, requiring no second anesthetization of the patient, and the wound remaining open behind facilitates approach to the middle ear for treatment and inspection during the healing period. The end results, so far as cosmetic effects are concerned, are as good as when the flap is primarily sutured, provided care be taken to prevent the edges of the skin from turning in during the progress of healing.

To summarize the steps of the radical operation:

1. After the antrum has been opened and the evisceration of the tympano-mastoid cells has been completed, the bridge of bone arching the tympanic ring is broken through from a point as near the middle of the superficies meatus as it is possible to go, nearing the zygomatic root rather than inclining away from it.

2. Granulations and detritus are removed from the tympanic cavity, including diseased ossicles.

3. Careful curettage of the Eustachian orifice.

4. Careful sponging of the floor of the tympanic cavity to identify dehiscences, and if such are found absent, then curettage of the floor. When dehiscences are identified, diseased tissue must be removed through sponging or by the use of thumb forceps.

5. Cutting the Panse flap and tamponing the edges of this flap in position through the external ear, packing the post-auricular wound with one strip of gauze, and finally placing the outside dressings in position.

Faults of Technic: The osseous canal being less and less evident the younger the subject, the part played by the squamous must be remembered. As the result of the canal being attached at its roof to the superficies meatus, the auricle is situated high relative to the membrana tympani, and a common fault is failure to detach

the canal roof from the bony wall, and because of this the cranial cavity is unnecessarily opened. I have found a false opening into the cranium, in a case under observation within the year, made because of this fault, and the operator in this case failed to uncover the antrum.

Contrary to the case in the adult, where the antrum is usually higher than is supposed, in the child it will be lower if the primary incision is not properly made and the soft canal carefully separated from the superficies meatus. Oppenheimer (l. c.), calls attention to the fact that the squama, instead of occupying an almost vertical position, is inclined, making an acute angle with a horizontal plane, and causing the linea temporalis to overhang the external auditory canal. This overhang might be mistaken for the temporal ridge and the operator, instead of opening into the antrum, will open the cranial cavity. Through gentle persistence in retracing soft parts until the membrana tympani is recognized the unusual position of this ridge will be identified and this fault of techinc averted. Many operators make the suprameatal triangle their guide. In the child neither this, nor Henle's spine, if it be present, can be depended upon. The position of the antrum is so different at different ages that these landmarks can no longer be accepted as reliable guides in the surgery of this region in the young.

In closing this paper I wish to call attention to the red-marrow bone character of the cells in the young subject. Very often, after the central focus of infection has been removed by the operator, he will proceed to eviscerate and enlarge the bony cavity indefinitely, expecting to find hard bone; but when it is remembered that in many cases the developing temporal bone of the child is soft and of red-marrow, and easily broken down with the curette, such a mistake of technic can hardly happen.

BIBLIOGRAPHY.

1. Gruber. Untersuchungen des Aeussern Gehörganges mit besonderer Rücksicht auf die praktische Ohrenheilkunde. *Monatschr. f. Ohrenh.* 1868, 6.
2. Haug. Die Krankeiten des Ohres in ihrer Beziehung zu den Allgemeinerkrankungen. Wien und Leipzig, 1893.
3. Gomperz. *Centralb., f. d. ges. Therap.,* 1894, page 299.
4. Otto Laubi. Methode und Resultate der Ohren-untersuchungen von 22894 Schülern der ersten Primarklasse der Stadt Zürich. *Correspondenzblatt, f. Schweiz., Aerzte,* XIII, 1903.
5. Barschmann. Frequency and Importance of Ear Disease in School Children. Jahrsammlungen Kinderaertze. Gesellschaft zu Moskau, Feb., 1904.
6. Buerkner. Statement in the Transactions of the 10th Intern. Medical Congress, Berlin.

7. Bezold. Statistischer Bericht über die in den Jahren, 1893-96, inclusive, behandelten Ohrenkranken. *Ztschr. f. Ohrenh.,* Vol. 32, 1898.
8. Barth. Otitis Media in Infancy. *Ztschr. f. Ohrenh.,* Vol. 32, 1898.
9. Ponfick. Ueber die Allgemein-pathologischen Beziehungen der Mittelohr-Erkrankungen im frühen Kindesalter. *Berl. klin. Wchnschr.,* No. 38-41, 1897.
10. Barbarin. *These de Paris,* 1902.
11. Rasch. Ueber die Haeufigkeit und Bedeutung von Mittelohrentzündungen bei kleinen kranken Kindern. *Jahrb. f. Kinderh.,* No. XVIV, 1894.
12. Kassel. *Charité-Ann.* No. XVIII, page 497.
13. Hartmann. *Deutche med. Wchnsch.,* No. 26, 1897.
14. Henrici. Weitere Erfahrungen über die Tuberkulose des Warzenfortsatzes im Kindesalter. *Zthchr. f. Ohrenh.,* Jan., 1906.
15. Haike. Tuberkulöse Ohrenerkrankungen im Saeuglingsalter. *Deutche. med. Wchnschr.,* No. 24, 1905.
16. Korned. Die Eiterigen Erkrankungen des Schlafenbeins (Wiesbaden, 1899).
17. Ostman. *Arch. f. Ohrenh.,* Vol. LV.
18. Holt. Diseases of Infancy and Childhood, 1897.
19. Bezold. *Arch. f. Ohrenh.,* No. 21, 1884.
20. Jansen. Die Entzündungen des Mittelohrs und ihre Behandlung. *Deutch. klin. chir. Vorlesungen,* 1905.
21. Haug. Die Krankheiten des Ohres in ihren Beziehungen zu den Allgemeinerkrankungen, 1893.
22. Heurner. Scharlachfieber. Deutch Klinik (*Kinderkrankheiten*), 1905.
23. Burchartd-Merian, *Volkmann's klin. Vortraege,* No. 182, 1880 (Scharlach im Gehörorgan).
24. Wendt. Gehörorgan und Nasenrachenraum bei Variola. *Arch. f. Heilk.,* Vol. XIII, page 118, No. 414, 1872.
25. Goerke and Brieger. *Arch. f. Laryngol.,* Vol. XII, No. 2, 1902.
26. Barth. Pharyngeal Tonsil and Ear. *Monatschr. f. Ohrenh.,* No. 11, 1897.
27. Kerley. Acute Middle Ear Disease in Childhood. *Arch. f. Paediat.,* Oct., 1904.
28. Lartigue and Nicoll. A Study of the Pharyngeal Lymphoid Tissue (Adenoids) with Special Reference to Primary Tuberculosis of the Pharyngeal Tonsils. *Am. Jour. Sc.,* June, 1902.
29. Zappert. Ueber einige dem Kindesalter eigentümliche Erkrankungen der Nase und des Rachens. *Deutche. Klinik.,* Vol., VII, 1905 (Kinderkrankheiten).
30. Eitelberg. Ein Beitrag zur Bougierung der Ohrentrompete, *Ztsch, f. Ohrenh.,* Vol. XIII, page 132, 1884.
31. Schengelidze. *Arch. f. Kinderh.,* 1891.
32. Fabricinab Aquapendente. De visione Etaudite, 1600.
33. Link. Citation of Older Literature, Handbuch der Ohrenheilkunde.
34. V. Troeltsch. *Verhandl. d. prys. med. Gesellsch. zu Wurzburg,* IX, 1859; Lehrbuch der Ohrenheilkunde (all editions).
35. Wendt. *Arch. f. Ohrenh,* XIV, 1873.
36. Boeke. *Jahrb. f. Kinder.,* 1876.
37. Weiss. Beitraege zur Path., Anat. und zur allg. Pathologie, L. E. Ziegler, vol. 27, 1900.
38. Kutscharianz. *Arch. f. Ohrenh,* X. 1876.
39. Preysing. Otitis media des Saeuglings, Wiesbaden, 1905.
40. Gomperz. Path. u. Therapie der Mittelohrentzündungen in Saeuglingsalter, Wien, 1906.
41. Urbantschitsch. Handbuch der Ohrenheilkunde.

42. Bryant. Anatomy of the Child's Ear emphasizing points of practical importance. *Arch. of Otol.,* Vol. XXXIV.
43. Dench. *Arch. of Otol,* Vol. XXVII, Page 297.
44. Gruber. *Monatschr. f. Ohrenh.* 1900.
45. Koerner. Die Otitischen Erkrankungen des Hirns, der Hirnhaeute, und der Blutleiter. Wiesbaden, 1902.
46. Barbarin. *These de Paris,* 1902.
47. Symington. The External Auditory Meatus in the Child. Jour. *Anat. and Physiol.,* Vol. XIX, 1885, Page 111.
48. Kopetzky. Surgery of the Tympano-mastoid Region of the Infant and Young Child. *Am. Jour. Surg.,* October, 1906.
49. Baginsky. Ueber Gewisse Eigenartigkeiten der Ohrerkrankungen der Kinder. *Berl. klin, Wchnschr.* Feb. 10, 1902.
50. Zaufal. *Prager med. Wchnschr.,* 1887, 1888, 1889.
51. Netter. *Arch. gen. de med.,* April, 1887.
52. Weichselbaum. *Monatschri. f. Ohrenh.,* 8-9, 1888.
53. Gradenigo and Penzo. Bakterial Beobachtungen über den Inhalt der Trommelhöhle im Kadavar von Neugeborenen und Saeuglingen. *Ztschr. f. Ohrenh.* XXI., 1890.
54. Brieger. *Klin. Beitr. z. Ohrenh.* F. Bergman, 1896.
55. Preysing. *Centralb. f. Bacteriol.,* Vol. XXV, Page 655, 1899.
56. Kossel. Ueber Mittelohrentzündung bei Saeuglingen. *Charite-Ann.,* XVIII, 1893.
57. Haberman. Ueber die tuberkulöse Infektion des Mittleohres. *Ztschr. f. Heilk.,* Prag. 1885.
58. Flesch. Zur Aetiologie der Ohreneit. im früh. Kindesalter. *Berlin.* klin. *Wchnschr.* No. 48, Pake 1234, 1892.
59. Held. Vincent's Bacillus and Spirillum, etc., the causal agent of chronic suppurations, etc. *Post-Graduate Med. Jour.,* September, 1906.
60. Weiss. Zur Aetiologie und Path. der Otitis Media im Saeuglinsalter. (*Beitrag zur path. Anat. und Algemein Path.,* Vol. 27.)
61. Scheniglidzé. Inaugural Dissertation. St. Petersburg, 1900.
62. Streckeisen. Bericht über die Kindsep. v. Bassel. 1864.
63. Schwartz. *Jour. f. Geburtshilfe-Frauenzimmer und Kinderkrankheiten,* Vol. V., Part 1, Page 160.
64. Melsner. Lehrbuch der Kinderheilkunde, Leipzig, 1828.
65. Helfft. Jour. f. Kinderk., Vol. IX, Page 401, 1847.
66. Hauner. Beitrag zur Padiatrik Bd. I. Berlin, 1863.
67. Hartmann. *Deutsche med. Wchnschr.,* 1894.
68. Schengelidzé. Die Eitrigen Entzündungen bei Brustkindern, Inaug. Dissert. Strassburg.
Schengelidze. *Arch f. Kinderh.,* Vol. XXX, 1901.
69. Goppert. *Jahrb. f. Kinderh.,* 1897.
70. Goppert. Vol. 44, 45, No. 3. *Jahrb. f. Kinder.*
71. Ponfick. *Berlin klin. Wchnschr.,* 1897, Page 817.
72. Hartmann. *Ztschr. f. Ohrenh.,* Vol. 34, 1898.
73. Jacobi. *Arch. Otol.,* Vol. XXXIV, No. 2.
74. Homer and Laitinen. Die Wirkungen von Streptokokken und ihren Toxinen. *Ziegler's Beitrag,* Vol. 25, Heft 1.
75. Gorden A. *Knyvett, med. Chron.,* Sept., 1905.
76. Scheibe. *Munch. med. Wchnschr.* No. 21, 1900.
77. Ostman. Die Prophylaxis des Ohres bei den Akuten Infektions-Krankheiten. *Munch. med. Wchnschr.,* 1905.
78. Weiss. *Wiener med. Wchnschr.* No. 52, 1900.
79. Love. *Glasgow Med. Jour.,* April, 1906.
80. Kopetzky. Acute Purulent Otitis and Mastoiditis treated by means of artificially induced Hyperaemia, according to the method of Bier with report of cases. *Arch. Otol.,* Vol. XXXV, No. 4, 1906.
81. S. Oppenheimer. Surgery of the Middle Ear and Mastoid. Blakiston, 1906.
82. McEwen. Pyogenic Disease of the Brain and Spinal Cord.

PAPER:

TUBERCULOSIS OF THE MOUTH.

ROBERT LEVY, M. D.

Tuberculous lesions of the mouth include those involving the lips, cheeks, gums, hard palate, soft palate, tongue, teeth and alveolar process. Tuberculosis of the pharynx is not included in this paper, although it is difficult to draw the dividing line. So large a subject as tuberculosis of the tonsils forms a sub-division of pharyngeal tuberculosis and is also omitted from this discussion, although it will be necessary for purposes of comparison and illustration to touch upon all lesions of tubercular character ordinarily seen by what is known as pharyngoscopy. It is comparatively rare to see isolated lesions involving but one of the structures included in the mouth, and it is the author's belief that such rare cases form a class by themselves, possessing the same general characteristics that other tuberculosis lesions of the upper air passages do, but marking a distinct difference in course and prognosis.

A careful review of over 200 references, abstracts of which will appear in a subsequent paper, impresses one with the rather large number of tuberculous lesions of the mouth which have, from the time of Thomae's article,[1] 1839, been reported. The largest number, however, have appeared within comparatively recent years and I wish to mention especially the early articles of Bosworth,[2] DeBlois,[3] Delavan.[4] Yet when one analizes the extensive literature, he must be struck by the general inaccuracy in diagnosis and the lack of appreciation of district variations and differences in these lesions; differences which are of special importance in etiology and prognosis.

FORMS.

Tuberculous lesions may manifest themselves in different forms, i. e., according to pathologic changes, according to mode of development and according to their clinical course.

First; the various pathologic alterations seen are nodular infiltration, superficial ulceration, deep ulceration, perforating ulceration, necrosis of bone, chronic abscess and tumor.

Second; according to the mode of development, we have two forms, "endogenous" and "ectogenous" (Grünwald),[5] which corresponds to the general classification of Holländer[6] into "descending" and "ascending."

Third; according to clinical course, we may have "malignant" and "benign."

Clinical observation confirms this classification. The "extogenous" or "ascending" form, that which may be designated as the inoculation variety or purely local, represents the less active, sluggish or "benign" type, while the "endogenous" or "descending" variety, that which represents infection through blood and lymph streams, through miliary deposits or infection from within, corresponds to the more active, virulent, malignant type.

ETIOLOGY.

It is a well established fact that tuberculosis of the upper air passages, and particularly of the mouth and pharynx, is more commonly found in males than females. This leads to the question of how far local irritation of the mouth and possibly slight injury of the mucous membrane enter into causation of tuberculosis. This also raises the question as to the existence of primary tuberculosis of these structures. There are innumerable cases upon record in which the only demonstrable lesion is that localized in the mouth and especially upon the tongue. In these cases, the history of ulceration following a slight injury to the mucous membrane, which although nothing more than an abrasion to begin with, refused to heal, should be fairly conclusive proof that the abraded surface was an easy portal of infection in which tubercle bacilli became lodged, the characteristic tubercular process following. On the other hand, assuming that there is a necessary vulnerability without which tubercle bacilli may be innocuous, leads to the belief that there already exists in individuals developing such a localized tuberculosis, another and primary focus of infection. Auguy[7], 1895, and others since then have stated that the most frequent mode of infection is through the blood current. Walsham[8] has shown the importance of the lymphatic system in the development of tuberculosis especially with regard to the frequency with which infection may be conveyed to the bronchial glands from above, through the adenoid tissue in the throat, including the pharyngeal and faucial tonsils. It has been unquestionably demonstrated that tuberculous infection may invade the system through the tonsils with-

out producing any alteration in the tonsils themselves. It has also been shown by Cook[3] that the teeth, especially when diseased, form excellent foci through which infection may be carried to the adjacent lymphatic structures. The question, therefore, becomes an extremely complicated one, especially when we remember that post mortem examinations have revealed the presence of foci of tuberculosis in the lungs which were not suspected and could not have been demonstrated ante mortem. It seems reasonable to conclude that, although local or systemic causes exist, infection may descend through the lymphatic system to the bronchial glands, invade surrounding structures and be carried by the blood current to such parts as the tongue, the lips, the gums or the hard palate, and that additional local irritation or trauma, however slight, may determine the outward manifestation of tuberculosis in these regions. Ragged, sharp or decayed teeth, extraction of teeth, poorly fitting dentures, biting the tongue, injury from pins or other sharp bodies in the mouth constitute the more common local cause. In fact, when we consider that the mucous membrane of the mouth, although more or less constantly bathed in tubercle bacilli laden sputum in tuberculous individuals, develops tuberculous lesions but rarely, one must admit that there exists a special protective agency. This protective agency obtains so long as the surface of the mucous membrane is not altered either by traumatism or pathologic changes. Once this protective agency is in a measure diminished, local tuberculosis may manifest itself. The more frequently one sees tuberculosis of a given organ, the more reasonable it is to presume that the infection of that structure came about through the blood or the lymph current. The less frequently certain structures are involved, the more reasonable to suppose that some local cause must operate to overcome its invulnerability. This thought is supported in the classification of Grünwald, Walsham and others as well as in the clinical history. It is a recognized fact that the so-called primary lesions of the mouth are more sluggish and less malignant than the secondary varieties, the latter developing through blood and lymph current in an organism abundantly attacked by the tuberculous process. This though is further borne out by the slow and comparatively non-malignant affections of the gums, the hard palate, the lips, the cheeks and the tongue, and the rapidly progressing, actively malignant course of tuberculosis of the pharynx and tonsils.

SUBJECTIVE SYMPTOMS.

Tuberculous lesions involving the tongue, the lips, the gums, may exist for some time before the patient is aware of their presence. Even ulcerating lesions may develop to a considerable extent before attracting attention. In this respect, these lesions differ from those involving the pharynx and larynx. The earliest manifestation is a sense of slight soreness or burning, marked particularly when the diseased surface is irritated by contact with food. Soreness may become decided pain, more especially when the lesions involve the tongue. When seen upon the hard palate or gums the pain rarely becomes severe, differing from the usual intense pain of tuberculous ulcers. There is localized swelling and slight increase in the secretion of the parts, which becomes viscid, grayish white or dirty. A slight odor is often apparent, although rarely becoming offensive. Even when the ulceration has gone on to marked necrosis of the underlying structures, the odor that is at time discernable may be attributed to the general cachexia of the individual rather than to the local lesions. Glandular involvement may or may not be present. I have seen it in very early lesions and have failed to discover any enlargement of cervical glands in cases far advanced. When it does exist it is rarely painful. In tuberculosis of the tongue, lips, gums or palate, the general symptoms of the patient are comparatively slight except in those cases which develop in the course of severe general infection, such as is found in a miliary process. These severe cases are usually marked by the rapid development of lesions extending to the soft palate, the tonsils and the surrounding pharyngeal structures when dysphagia and odynophagia of severe type, high temperature, rapid pulse, marked emaciation and exhaustion may be added to the symptoms.

OBJECTIVE SYMPTOMS.

Excluding those isolated instances of tumor and abscess,[10] there is apparent to the most casual observer a definitely uniform local lesion. The general appearance is that of a pale, superficial ulceration without inflammatory areola, edges irregular in outline and beveled or undermined, tending to spread latterly, not deeply. A viscid, dirty white secretion covers the ulceration, which when cleaned away brings to view a more or less nodular appearance. Scattered irregularly over the surface of the ulceration and upon its margins are seen small red, soft granulations, interspersed with pin head spots of yellow or gray. These yellow spots, the spots of

FIGURE I. CASE I.

FIGURE II. CASE I.

FIGURE III. CASE II.

FIGURE IV. CASE II.

FIGURE V. CASE III.

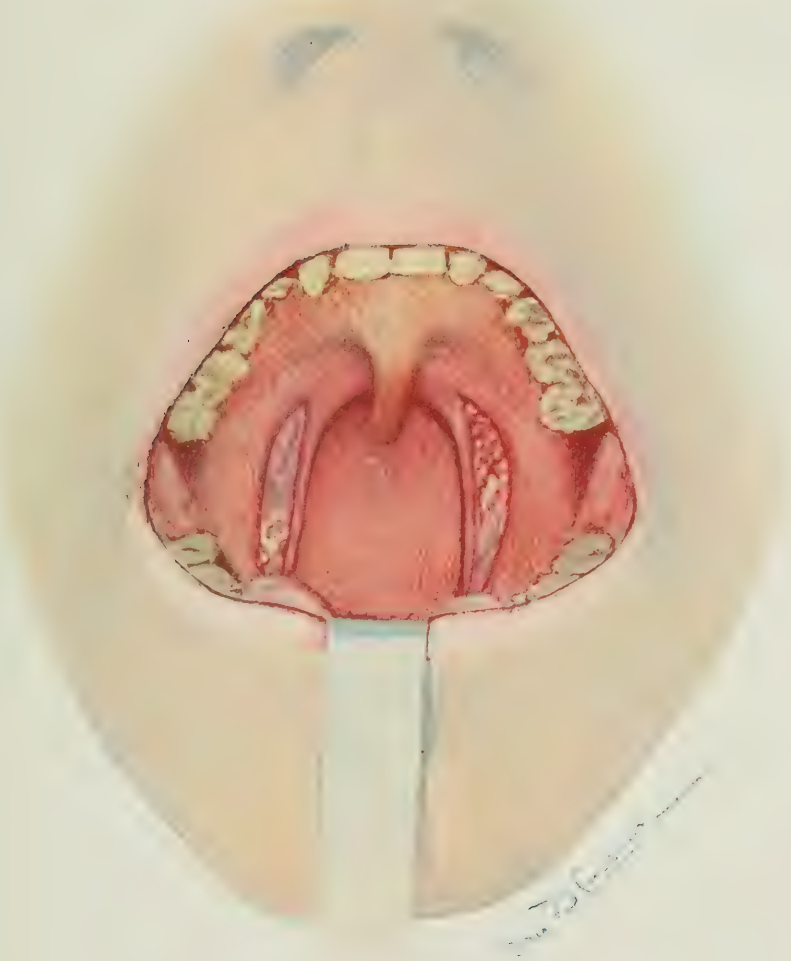


FIGURE VI. CASE IV.

FIGURE VII. CASE V.

FIGURE VIII. CASE VI.

FIGURE IX. CASE VII.

FIGURE X. CASE VIII.

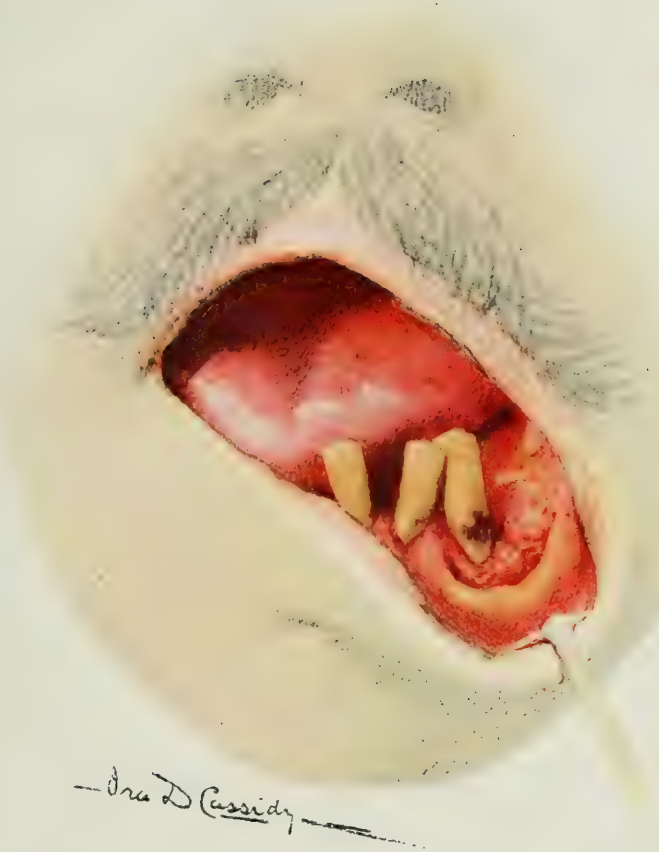


FIGURE XI. CASE VIII.

Trelat,[11] may be seen also upon the mucous membrane adjacent to the ulceration.

Case I (Figures 1 and 2) demonstrates very early as well as moderately advanced tuberculosis of the gums. This patient, aged 26, was first seen in December, 1906, having been referred by Dr Bergtold. He has had pulmonary tuberculosis one year. In August, 1906, the gums became sore. There was also some soreness in the nose and larynx, the latter inducing painful and difficult swallowing. A typical tuberculous ulcer was seen upon the left surface of the nasal septum. The larynx presented pale infiltration of both arytenoids with numerous small ulcers and grayish deposits. The gums over the first bicuspid on the right side of the upper jaw and over the first and second bicuspid right side, lower jaw, were seen to be covered with small redish nodules, very superficial ulcers and a few pin head yellowish spots. The typical tuberculous appearance of a very early lesion was readily demonstrated. Upon the lingual surface of the gums of the first and second molars, lower jaw left side, more advanced lesion was seen, possessing, however, the same characteristic nodular superficial ulceration with redish and yellowish spots. Although typical in appearance, repeated and thorough examination of curettings of these lesions by Dr. Todd failed to demonstrate the presence of tubercle bacilli.

The margins of the ulcerations are rarely indurated. Upon the tongue, however, a slight induration may be felt and especially where healing has occurred, leaving the organ markedly fissured. The character of these indurations is a fibrosis, which has occurred in the healing process.

Case II (Figures 3 and 4) shows a typical case of tuberculosis of the tongue. Figure 1 illustrates the fissured appearance which has resulted in the healing process. The induration on the margin of these fissures being marked. Figure 2 shows an active lesion at the tip of the tongue, presenting all the usual characteristics of tuberculous ulceration. This case was that of a man aged 35, who has had tuberculosis for seven years. He came to Colorado immediately upon development of the trouble. Eleven years ago or four years before pulmonary tuberculosis was detected, he had a small superficial ulcer upon the dorsum of the tongue, which refused to heal. Subsequently similar ulcerations on the dorsum developed and continued active for an indefinite period. The patient cannot state exactly when healing began. One year ago the ulceration upon

the tip developed, which was extremely painful, a symptom which did not obtain in the ulceration upon the dorsum. When seen at the National Jewish Hospital for Consumptives in December, 1906, the healing process upon the dorsum of the tongue was complete with the exception of two small lesions. The tip of the tongue, however, was in a condition of active ulceration. Careful curettings from the dorsal as well as tip ulcers, showed tubercle bacilli in intimate relation with the epithelium cells. At the present time, May 23rd, 1907, the patient's general condition is slowly failing, the ulceration upon the tongue, both dorsal and tip, are perfectly cicatrized.

When the disease extends to the soft palate, the anterior or posterior pillars, the uvula or the tonsils, a somewhat different picture presents itself. The very earliest appearance is a marked pallor which is heightened by an edematous swelling. This edema may be extremely slight, but it gives to the structure a characteristic appearance. Very careful observation will reveal the presence of numerous yellow or grayish pin head spots just under the surface of these pale, edematous structures.

Case III. (Figure 5.) This condition is typically illustrated by the following cases. C. Mc., male, aged 28, referred to me Oct. 2nd, 1906, by Dr. S. Solis Cohen of Philadelphia. The patient had slight pulmonary involvement and a tuberculous ulcer of the left arytenoid and vocal band. These lesions had improved under Dr. Cohen's treatment, the improvement continuing after coming to Colorado. On the 27th of February, 1907, both tonsils were swollen, pale and dotted with a few small grayish deposits. The posterior pillars were slightly edematous. The patient's general condition was failing. A small piece of tonsillar tissue was removed and sections made by Dr. Todd, who reported numerous miliary tubercles, giant cells and caseous centers. A few tubercle bacilli were demonstrated at the edges of the caseous areas.

Case IV. (Figure 6.) Male, aged 24, referred by Dr. Levitt of New York. This patient was presented before the section of Laryngology at the New York Academy of Medicine by Dr. Emil Mayer, Feb. 27th, 1907, demonstrating a possible primary lupus of the larynx. Upon examining the patient March 9th, there were found slight pulmonary involvement, swollen epiglottis, which was red and covered with pin head grayish tubercles and notched in the center, probably from a section removed. The aryepiglottic folds were swollen, pale, edematous and covered with grayish de-

posits. The arytenoids were moderately swollen and pale. The patient's condition rapidly grew worse and upon April 9th, there was discovered on the right tonsil a whitish deposit. The left tonsil presented a small, irregular, nodular ulceration with whitish imbedded masses. Scrapings from both tonsils were submitted to Dr. Todd, who reported as follows: "The preparation from the left tonsil shows numerous cells, the structure of which cannot be clearly seen. Careful search shows very few tubercle bacilli scattered among them. The cover glass preparation from the right tonsil contains many squamous epithelial cells and a few other cells which cannot be made out distinctly, and some degenerated material. No tubercle bacilli can be found. Figure 6 shows distinct difference between the two tonsils, the right presenting an exudate upon its surface, while the left shows the typical tubercular appearance. The difference in the two tonsils as demonstrated clinically is borne out by the laboratory report.

Following the deposit of tubercles ulceration rapidly develops, the yellowish spots breaking down and coalescing in an irregular manner, giving to the surface of the ulceration the characteristic worm eaten or mouse nibbled appearance. When the ulcerations become deep, as they sometimes do, they still retain upon their margins the characteristic tubercular appearance. This is demonstrated by the following case:

Case V. (Figure 7.) Male, aged 44, advanced tuberculosis of the lungs and larynx of two years' standing. Three weeks before I saw this case the gums became sore and an ulcer developed upon the upper jaw. This seemed to be the result of loose and decayed teeth, which were plainly apparent. The ulceration is a typical deep tubercular ulceration extending to the alveolar process, which is itself necrosed, showing an exposed tooth root. A section of the margin of the ulcer was removed and the laboratory report gives the following: Granulation tissue and a few well defined tubercles with caseous centers; small numbers of tubercle bacilli scattered about the periphery of the tubercles.

Extensive ulcerations involving the soft palate and posterior structures are illustrated by:

Case VI. (Figure 8.) Male, aged 40, advanced tuberculosis of the lungs, sore throat at times during the past seven years. For three weeks past there has been painful and difficult swallowing. Examination of the pharynx shows extensive mouse eaten appearance involving soft palate, both pillars of the fauces, tonsils, uvula

and posterior wall of the pharynx downwards to the left side of the larynx. The ulceration extending forward as far as the junction of the soft with the hard palate.

DIAGNOSIS.

The lack of scientific accuracy so frequently displayed in the diagnosis of tuberculosis of the mouth and pharynx is worthy of note. Any or every ulceration of the mucous membrane of the mouth or pharynx in a tuberculous individual is not necessarily tuberculous. It is feared that this has too often been believed and that it may be the reason so many cases of cure have been reported.

Illustrating this, I present:

Case VII. (Figure 9.) H. T., aged 32, tuberculosis of the lungs, one year. Ulceration on the under surface of the tip of the tongue for six months. These ulcerations are two in number and are not painful. They present a marked red, inflammatory areola, are irregular in outline and show nowhere any of the characteristic grayish or yellow spots, or redish granulations. They are evidently due to contact with the sharp lower incisors, aggravated probably by more or less persistent coughing. When last seen, May 19th, the lower ulceration had entirely healed without treatment.

The diagnosis is certain when there is a pale ulceration, without inflammatory areola, superficial, worm eaten in appearance, dotted with redish pin head elevations and having small yellow or grayish spots scattered throughout. An early diagnosis when the soft palate or tonsils are involved may be assumed when there appears an extremely pale, slightly edematous mucous membrane with small, sub-mucous, pin head, yellow spots.

These diagnostic features, however, are not always present, although they do exist in the vast majority of instances. In all cases the diagnosis should be confirmed by the microscopic findings, and in obscure cases this and the inoculation of Guinea pigs are the only positive tests. The finding of giant cells and caseous material together in sections is almost certain proof of tuberculosis.

In case of the ulcers, the laboratory confirms the diagnosis, according to Dr. J. C. Todd of the Pathological Laboratory of the Denver and Gross College of Medicine, in two ways:

1. Detection of tubercle bacilli, (a) by the microscope, or (b) by inoculation of Guinea pigs.

2. Detection of the histological structures of tubercle in stained sections.

(1) Detection of the bacilli:

(a) Owing to possible presence of tubercle bacilli in the mouth, simple examination of swabs from the surface of the ulcer is of little value. The surface of the ulcer should be thoroughly cleansed and then under cocaine, curetted; or a piece of sufficient size for sections should be exercised. Curettings are to be thoroughly rubbed between slides or cover glasses until the cells are sufficiently dissociated to allow thin smears. The presence of tubercle bacilli in the smears may be taken as proof of tuberculosis, provided the surface of the ulcer was well cleansed, and this is made absolutely sure by finding the bacilli within the small clumps of cells which the rubbing between slides failed to separate.

When a piece of sufficient size can be excised, it should be sectioned and stained for tubercle bacilli. Tubercle bacilli are sometimes abundant even when the structure of the tubercle is very doubtful.

(b) Inoculation of Guinea pigs is resorted to only when other means fail.

(2) The histological structure of the miliary tubercle can generally be found in portions of tissue which have been sectioned and stained. Recognition of the tubercle depends upon the presence and, especially, the arrangement of certain structures; no one structure is diagnostic in itself, although its presence may be very suggestive. However, the presence of giant cells and caseation *together* may generally be accepted as proof when the structure is not otherwise typical and only as forming a part of a tubercle.

Grünwald[12] has shown how difficult it is to find tubercle bacilli in all tuberculous ulcerations. Hajek[13] has also called attention to this fact in tuberculosis of the nose, therefore, one cannot place all of his reliance upon the presence of these organisms. With few exceptions will one fail to find them present if sufficiently long and accurate search be made. They are usually few in number and scattered and are rarely absent in typical lesions. In the cases presented under Figures 1 and 2, the local lesions were absolutely typical in appearance, but in spite of most thorough and repeated search, tubercle bacilli were not found.

PROGNOSIS.

When the affection involves the structures anterior to the soft palate including the tongue, gums, lips, cheek, hard palate, the prognosis so far as the general lesion is concerned is of comparatively little importance.

Case VIII. (Figures 10 and 11.) V. G. L., aged 58, has been ill with tuberculosis of the lungs for over fifteen years. Slight ulceration of the hard palate and gums were noticed in October, 1906. These lesions have been comparatively free from pain. There is, however, marked soreness and discomfort especially upon eating. Figure 10 shows a typical and characteristic tuberculous ulceration of the hard palate with beveled edges and nodular base. Figure 11 shows more advanced tuberculosis of the gums, rather deeper than usual with undermined edges, especially inferiorily. Careful curettings from both of these ulcers were examined by Dr. Todd, who easily demonstrated tubercle bacilli, but the entire absence of all other bacteria showed the thoroughness with which the parts were cleansed and proved conclusively that the tubercle bacilli were obtained from the surface of the ulcerations themselves and were not the result of contamination.

Tuberculous ulcerations rarely heal, but at the same time their progress is extremely slow. They are the source of but little discomfort and have no special bearing upon the course of an associated general or pulmonary tuberculosis except in so far as they indicate involvement of a small amount of additional tissue. Some of these cases have been said to heal spontaneously, others to have been cured by excision or cauterization. Unquestionably a few rare instances of such cures may be accepted as authentic, such cases representing that comparatively benign form described as "ectogenous' or "ascending." Bernheim[14] states that in buccopharyngeal tuberculosis a cure is the rule. Gleason[15] also states that the "Prognosis as regards healing is favorable." So firmly convinced am I that these statements are erroneous that I venture to question the diagnosis.

When tuberculous lesions involve the soft palate, uvula, the tonsils, anterior pillars and the structures posteriorily, the prognosis is of very much more significance. Not only is the prognosis of the local lesion absolutely unfavorable, but their effect upon the general condition of the patient is so deleterious that one can predict decline and an early and fatal termination. Lesions here are positive evidence of rapidly disseminating miliary tuberculosis.

Two exceptions to the above statements should be made. First, when the lesion involves the tongue the prognosis may be quite as unfavorable as when it involves the pharyngeal structures and second, comparatively benign lesion may exist in the posterior wall of the pharynx.

TREATMENT.

This consists in palliative and curative and should be both local and general. Palliative treatment is directed to the relief of pain. The local application of cocaine or of powdered orthoform are the most valuable remedies. Pain may also be mitigated by curetting and cauterizing, on the theory that the pain is due not so much to the exposed nerves as to the development of small neuromata upon the exposed nerve filaments.

Curative ulcers which involve a small portion of the tip or margin of the tongue, or localized tumors have been removed by more or less extensive, radical excisions. Temporary healing may be brought about by thorough cauterization followed by frictional rubbings with lactic acid or formalin. Galvano cautery has been of some avail and particularly have I seen early tubercular ulcers of the tonsils, lips and tongue healed under this treatment. Usually these healed ulcers reappear or there develop others adjacent to them. In spite of the generally accepted ultimate fatal outcome of these cases, one should not neglect an attempt at cure, and therefore, in addition to local treatment the usual attention should be paid to the patient's general welfare. Rest is of prime consideration, owing to the frequent presence of high temperature. The patient's nutrition should be maintained to the highest possible standard by the administration of large quantities of easily digested, nutritious food. When much pain exists this becomes a question of serious moment and local anaesthetics should be abundantly applied. Feeding by means of esophageal tubes cannot be recommended, for the passage of such a tube is of as much discomfort to the patient as the act of swallowing.

BIBLIOGRAPHY.

1. Thomae, F. J. De induratione scrofulosa linguae et genarum observatio, 1839.
2. Bosworth. Trans. Am. Laryng. Ass'n. 1897.
3. DeBlois. Trans. Am. Laryng. Ass'n. 1884.
4. Delavan. Trans. Am. Laryng. Ass'n. 1886.
5. Gruenwald, Atlas Mouth, Pharynx and Nose, p. 126.
6. Hallonder. *Berl. klin., Wchnschr.,* June 4, 1906.
7. Auguy. De la tuberculose linguale. These de Paris 1895, Ref. *Intern. Centralblt. f. Laryng.,* etc., 1896, p. 212.
8. Walsham. Channels of infection in Tuberculosis 1905.
9. Cook, *Dental Review,* Feb. 1899.
10. Richardson. Trans. Am. Med. Ass'n. 1897 (Section Laryng.), p. 73.
11. Trelat. *Arch. gen. de med.,* Paris, 1870, Ref. *Centralblt. f. Laryngol.,* etc. Berger p. 295.
12. Gruenwald, Atlas Mouth, etc., p. 128.
13. Hajek. Heymans Handbuch.
14. Bernheim, Bucco-pharyngeal Tuberculosis, *Tribune Medical,* Oct. 3, 1900.
15. Gleason, Manual of Diseases of the Nose, Throat and Ear, 1907, p. 206.

PAPER:

THE ETIOLOGY OF TUBERCULOSIS OF THE UPPER RESPIRATORY TRACT.

GEORGE BACON WOOD, M. D.

The great factor in the etiology of the disease, tuberculosis, is infection with the specific germ, the tubercle bacillus. Tuberculosis of all organs has, of course, this common feature, and for the sake of conciseness the discussion of the etiology of tuberculosis of any individual organ or set of organs should not include the morphology and pathologic power of the tubercle bacillus, but rather be confined to the peculiar resistance which the organ under discussion offers to the invasion of this germ. As far as the nose and throat are concerned certain portions are peculiarly resistant to bacterial invasion, while other portions are comparatively easily invaded. This variation in resistance depends both on the anatomy and the physiology of the different parts.

When we consider that the large majority of individuals breathe through the nose, and that the air which is forced through the nasal passages has been practically filtered free from dust and bacteria by that organ, it seems remarkable that tuberculosis of the interior of the nose is so comparatively rare.

St. Clair Thomson and Hewlitt experimented with an apparatus which enabled them to collect the air as it came out through the choanæ. In a given quantity of laboratory air they found 29 mould spores and 9 bacteria; whereas, after the same quantity of air had passed through the nose it contained only 2 mould spores and no bacteria. This experiment was confirmed again and again. The question naturally arises, what becomes of those organisms which lodge in the nose as the result of this filtration? The same experimenters placed a pure culture of the Bacillus Prodigiosus on the septum well within the vestibule. A diminution in the number of bacteria could be noticed within fifteen minutes, and practically no trace of the bacteria could be detected after eighty min-

utes; and after two hours had elapsed in no case could any growth be obtained in cultures from the spot inoculated.

This opposition of the nasal mucous membrane to the growth of bacteria was also demonstrated by Malatto. Piaget declared that the nasal cavity, with the exception of the vestibules, both in man and animals, is nearly free from germs. He says that this asepsis is largely dependent upon a bactericidal action of the nasal mucus, and that this mucus is absolutely fatal to the anthrax bacillus, the diphtheritic bacillus, some forms of streptococcus and staphylococcus, the colon bacillus and some other germs. Inglauer, on the other hand, states that normal nasal mucus does contain a certain number of bacteria, but he admits that the nasal fossa rids itself of a large number of the bacteria inspired. Wurtz and Lermoyez, in a recently published research, again demonstrated that the nasal mucus is bactericidal to the anthrax bacillus and to other germs to a greater or less extent. They obtained the nasal mucus from normal individuals by placing bits of packing in the nasal fossæ, the fossæ having been previously washed out and cleansed. The mucus in the fresh state and also after it had undergone sterilization was mixed with bouillon containing anthrax bacilli, and even after three weeks of incubation no growth could be found, and inoculated guinea pigs gave negative results. Parker and Wright, however, found the nose sterile in only 6 out of 36 normal individuals, but the non-sterile cases gave very few colonies.

In summing up the evidence which I have just gone over, it would seem that the mucous membrane of the nasal fossæ exerts an inhibitory influence upon most bacteria, and that most probably this inhibitory influence is due to the bactericidal action of the nasal secretion. It must not, however, be supposed that the nasal mucus is equally antagonistic to all germs, and it concerns us especially to determine if possible its action on the tubercle bacillus.

Strauss, in 1894, found virulent tubercle bacilli in the nasal cavities of persons who, not having tuberculosis themselves, were in more or less constant attendance upon tuberculous patients. The dirt, solid particles and mucous contents of the nasal cavities were removed by sterile cotton swabs from twenty-nine persons, including hospital patients, internes, and patients suffering from chronic maladies other than tuberculosis. The inoculations made with the material thus obtained showed that about 40% of persons examined had virulent tubercle bacilli in their nasal cavities.

Freudenthal, in 1896, investigated the naso-pharynx of 133 patients, 52 of whom were tuberculous and 81 suffering from some other diseases. Tubercle bacilli were found in all of the 52 tuberculous patients, and in 9 out of the 81 non-tuberculous.

In 1900, Jones removed the dust and crusts from the nasal mucosa, making inoculation tests with the material thus obtained. Thirty-one animals were used. The majority died of some unknown infection, but three showed distinct tuberculosis.

Lake holds that the nose very seldom forms a mode of entrance for the tubercle bacillus into the body. Under his direction, Lucas made cover-glass preparations from the nasal cavities of 50 tuberculous patients, in all of whose sputum tubercle bacilli were present. In only one case were tubercle bacilli found, though 80% of these cases showed some form of organism.

Guinea pig inoculations made by myself from four persons who had been more or less in contact with tuberculous patients gave negative results. I obtained the material by swabbing the floor of the nasal cavities with sterile cotton-wrapped applicators, washing them off in sterile water, and inoculating this water into the abdominal cavities of guinea pigs.

In a certain per cent of persons undoubtedly virulent tubercle bacilli will be found in the nasal cavity, but that they stay there for any length of time is not probable, so that tuberculosis of the nasal fossæ should be a comparatively rare condition.

Knight says that up to 1901 there had been 108 cases of tuberculosis of the nose reported. Ernst Pasch in 1905 reports 17 cases coming under his own observation. Other sporadic cases have been reported since then, among them one by Onodi and Rossi, and one by Marcelli, both of which were supposed to be primary.

It is very likely that a large number of cases of tuberculosis of the nose are overlooked, but even then it is remarkable that so few cases have been reported. A case of primary tuberculosis of the nose would, on account of its infrequency, undoubtedly be published. Personally I have never seen a case of primary tuberculosis of the nasal fossæ, and though I have had a fairly large experience during the last three or four years in examining the nasal cavities of cases of pulmonary tuberculosis at the Phipps Institute, I have seen very few secondary involvements.

Summing up, it would seem that the nasal cavities are only rarely infected with the tubercle bacillus, probably because the time neces-

sary for the propagation of this slow-growing organism permits the cilia of the nose and the nasal secretions to remove it from the fossæ; but it is also very likely that the nasal secretions themselves are directly inhibitory to its growth.

Tuberculosis of the pharynx should be considered as practically identical with tuberculosis of its lymphoid tissue. I doubt if the stratified squamous pavement epithelium, washed continually with a downward flow of mucous, is ever infected by the tubercle bacillus, except through the lymph-follicles, the lateral folds of the pharynx or one of the four tonsils. Statistics on pharyngeal tuberculosis are of little value in confuting or supporting this statement, because the large majority of writers have not differentiated between an ulcer on the lymph-follicles or the lateral folds from one situated between these structures. If we include under the term "pharyngeal tuberculosis" lesions of its lymphatic tissue then tuberculosis of the pharynx is not very rare.

There is a great discrepancy in the reported percentages of secondary pharyngeal tuberculosis, while Levy reports 67 cases in 500 autopsies; Böcher reports only 12 cases in 2950; and Lublinski 5 in 1,600. However, the average percentage of all these cases would be 1.4%, which corresponds pretty closely with the 1% of Guttman.

Sokolowski in 1903 called attention to the frequency with which the lateral folds of the pharynx become tuberculous, and showed that the condition resembles very closely the so-called latent tuberculosis of the tonsils, and like this condition in the tonsils the disease can be diagnosed only after portions have been removed and put under the microscope. In 13 cases of tuberculosis which he examined, 8 showed involvement of the granular tissue of the pharynx or of the lateral folds, that is 61½ per cent.

Cordes speaks of pharyngitis lateralis as the formation of a new small elongated tonsil; meaning by this that hypertrophy of the lateral folds of the pharynx is accompanied not only by hyperplasia of the lymphoid tissue but also by the formation of veritable crypts and follicles. It would seem, therefore, that the discussion of the etiology of tuberculosis of the pharyngeal mucosa would best be included in the discussion of the etiology of tonsillar tuberculosis.

When we come to consider the comparative susceptibility of the various parts of the upper respiratory tract to tuberculosis,

preference for liability to infection must be given to the tonsillar tissues. Let us consider the evidence which justifies this proposition. The crypts, which are characteristic of all tonsillar tissue, afford a comparatively open door for microbic invasion. The lumen of the crypt acts almost as an incubator, and the peculiar disintegrated condition of the lining epithelium does not, like the stratified epithelium of the mouth, present a mechanical barrier. The penetration of bacteria into the tonsil parenchyma is prevented almost solely by the vital resistance of the living tissue, and if the pathogenic power of the invading germ is sufficient there is practically no hindrance to its entrance within the confines proper of the human body. Both research work and clinical evidence afford abundant proof of this theory, demonstrating the peculiar susceptibility of tonsillar tissue to infection, and the ease with which virulent micro-organisms may pass through its structures.

The results of all the research work done in relation to general microbic invasion through the tonsils show that in the tonsil more than anywhere else on the body surface, external and internal, the penetration of the invading germ depends not so much upon any mechanical barrier presented by the tissue as upon the relative virulence of the germ. Therefore, because the streptococcus can infect the tonsil it does not follow that the tubercle bacillus can do so, and as we are speaking now only of tuberculosis it is not necessary to go into the details of the work done in relation to general microbic infection through the tonsils.

In active pulmonary tuberculosis there is a certain dosage of tubercle bacilli spread fairly equally over the upper respiratory tract, excepting the nose and nasopharynx, and it is probable that the frequency of secondary infections of the various parts will give a fair index to their relative susceptibility. Permit me to quote from a paper published by myself in 1904. The tonsils of 136 cases of pulmonary tuberculosis were examined by seven different observers, and in 94, tuberculous involvement was diagnosed, that is 69 per cent. The diagnosis in these cases was made chiefly by histologic examination. In very advanced cases of pulmonary tuberculosis the tonsils almost never escape infection. In 9 cases reported in 1904, in which I made post-mortem examination, the tonsils in every one showed tubercle with giant cells, and in 38 cases examined during the past year I found histologic changes typical of tuberculosis in 35, and I believe

they would have been found in the remaining 3 if the whole of both tonsils had been sectioned.

This predilection of tuberculosis for the tonsils has been confirmed experimentally by Bamp, Ravenel, and later by myself, In experimenting on hogs, I found that the tonsils could be inoculated by a single swabbing with virulent bovine tubercle bacilli, but not once in the whole series of experiments was there found any lesions in the upper respiratory tract outside of the tonsils.

It would seem that all that is required to produce tuberculosis in the tonsil is to bring in contact with this organ a number of bacilli sufficient to overcome the tissue resistance. Tuberculosis of the tonsils should then be the most frequent form of primary tuberculosis, as both food and air must first pass over these structures before gaining access to the more interior organs, and I believe this is true. In a series of 1671 cases of hypertrophied tonsils, compiled from the published records of twenty-three authors, primary tonsillar tuberculosis was diagnosed in 88, that is 5.2 per cent. I found about the same proportion myself in perfectly unsuspected cases, where the tonsils had been removed by operation and were only casually examined. It would seem safe to assume that at least 5 per cent of children have tuberculosis of the tonsils.

Probably in the majority of cases on which these statistics were based the tonsils examined were removed because of their enlargement, and therefore one is hardly justified on that ground alone in giving 5 per cent as the frequency of tonsillar tuberculosis in all children. We must, however, remember that the size of the tonsil bears no relation to the liability of infection, except that its enlargement presents, as it were, a larger net to entrap the organisms. From my examination of tonsils from persons who had died from pulmonary tuberculosis, the hyperplastic tonsil is apparently more resistant to tuberculous infection than the small tonsil in which there was very little lymphatic tissue. This observation, however, is based upon too insufficient evidence to be accepted in any other light than a suggestive one.

While it is scarcely within the scope of this paper to treat with such a large and important subject as the method of infection in pulmonary tuberculosis, it may be allowable at this time to speak briefly concerning the liability to systemic infection from tuberculosis lesions in the tonsils. Cornet, in an elaborate series of

experiments believes he has shown that the tubercle bacillus is capable of producing disease in any organ to which the germ gains access. If a given part of the body is inoculated the lesion develops locally, and is generally promulgated through the lymphatics to the nearest set of lymph nodes. The bacillus is arrested in these nodes and cannot proceed further until destruction of this barrier has been accomplished. When this resistance has been overcome then the infection proceeds to the next chain, and finally via the lymphatics to the venous system. The tonsils may be described as differentiated lymph glands, but it is not probable that they possess the same filtering qualities as the lymph nodes. By rubbing a virulent culture of bovine tubercle bacilli on the surface of the tonsils of the hog, I once succeeded in finding the tubercle bacillus in the regionary lymph gland of the neck within five days after the inoculation, and no demonstrable change could be found in the tonsillar tissue. In this same series of experiments, I further found that histologic changes appeared as early in the cervical lymph node as in the tonsil. Further, we know from clinical experience how quickly the cervical nodes at the angle of the jaw become enlarged and tender during infection of the tonsil, and that for a long time this tenderness is confined to the first set of glands. Hence it is evident that tuberculosis of the tonsils would very likely show the same clinical course as that of tuberculosis of the cervical lymph nodes.

Recently, several articles have appeared concerning the possibility of infection of the apices of the pleura and of the bronchial and mediastinal lymph glands from the upper respiratory tract via the cervical lymph chains. Grober, in 1900, believed he succeeded by injecting the region of the tonsils in living animals with India ink in getting the pigment to travel via the cervical lymph glands to the pleural apices and to the bronchial lymph glands. Fleiner had previously stated that the supraclavicular glands were anatomically directly connected with the other cervical glands, that is the deep lateral chain, and were at the same time regionary to the pleural apices. The clinical support which Grober brings to substantiate his theory is chiefly that the statistics collected from five different observers show that in 14.3 per cent of cases with tuberculosis of the glands of the neck the pleura became involved. There is no doubt that Grober has done a great deal of valuable work in this connection, and from a mere perusal

of his paper he seems justified in his conclusion that infection of the pleura may take place via the cervical lymph glands.

During the last year, an article has been published by Beitzke, going over this same subject very carefully, making both anatomical studies and experimental researches, also giving the data furnished by the post-mortem on 55 children. He directly opposes Grober's theory, and criticises his work, believing that his results do not justify his conclusions. His work has been most carefully carried out, and I quote his conclusions.

"1. There exists no lymph vessel leading from the chain of cervical lymph glands to the bronchial glands.

"2. Tuberculous infection of the lungs from the cervical lymph glands can take place only through the lymphatic trunk and the venous system.

"3. This path of infection, at least in children, is without any practical import. The infection of the lungs, and hence the bronchial glands, in children as a rule takes place through aspiration of the tubercle bacillus into the bronchi; a descending cervical tuberculosis may be present incidently.

"4. The aspirated bacilli may be in the respired air but they come from the mouth where they have gained access by food or by contact."

During the past winter I started a series of anatomical studies along the same line but unfortunately have not yet been able to complete them. The results I have so far obtained, however, support entirely Beitzke's claim concerning the connection between the supraclavicular lymph glands and those higher in the neck. On the other hand, in children the arrangement and number of the lymph nodes in the neck is very irregular, and the pleural apices come into fairly close relation not only with the supraclavicular glands but also with the extreme lower portion of the greater vessels of the neck. The deep lateral chain of the neck extends downward along these vessels, and if there should be a node situated in the lower part of the neck, as sometimes occurs, it is conceivable that tuberculosis of this node may infect the pleural apices directly by continuity of structure. It would seem, therefore, possible that the pleural apices may in some few cases be infected by descending tuberculosis of the lymph glands of the neck, but for the tonsillar lesion to reach this distance there are a great many lymph nodes which must first be broken down and

overcome. However, Beitzke would probably be correct in the large majority of cases when he says that infection of the lungs may theoretically occur from the cervical lymph nodes but that it would almost always be a miliary lesion due to the entrance of the bacilli into the venous system via the jugular lymph trunk. Miliary infection of the lungs was the only pulmonary lesion in my experiments on hogs, and this occurred only after all the nodes of the cervical lymph chain were broken down.

Another very probable source of systemic infection from local disease of the tonsils, especially from the pharyngeal tonsil, may come from the disintegration or breaking down of the tonsillar tissue and diseased portions being swallowed and inoculating the gastro-intestinal tract or the mesentric glands. A lesion, however, to do this would not belong to the so-called latent type but must be ulcerative and hence recognizable.

To sum up these various conflicting views is somewhat difficult, but it seems to me that as a rule the clinical importance of a tuberculosis lesion in the tonsillar tissues of the throat is about the same as a local tuberculous lesion in any other non-vital portion of the body, which is separated from the internal organs by an intervening chain of lymph nodes.

The etiology of laryngeal tuberculosis has been so extensively discussed of late years that it is hardly worth while to enter upon this subject, especially as I know of no new facts to offer. Probably the best synopsis of the subject will be found in the last edition of Lake's Monograph on Laryngeal Phthisis. I would, however, like to call attention just to a few facts.

It is unquestionable that primary tuberculosis of the larynx has occurred though it is exceedingly rare. Aronsohn has collected 34 cases which he divided into three groups; in the first group, those in which post-mortem examination showed tuberculosis of the larynx without involvement of the lungs, only 3 have been reported; in the second group, there were 9 cases in which were found advanced or old disease of the larynx with recent infection of the lungs; and the last group, 2 cases in number, were based upon clinical examination only. In other words, there has been at the time of Aronsohn's paper only 3 cases reported in which the primary condition of the laryngeal lesion had been anything like proven. Lake says that the term "primary laryngeal tuberculosis" implies an original invasion of the larynx in the absence

of any pulmonary phthisis, and does not, necessarily, exclude the presence of some such lesion as a tuberculous adenitis or otitis. To me it would seem better that by the term "primary involvement" it should be understood that the organ under discussion is the first in the body to show any lesion, and that the lesion must be termed secondary if it follows disease of some other organ, no matter how small or insignificant that organ may be. It is easily conceivable that the larynx may be infected from a tuberculous otitis, and such lesions should be placed under the secondary laryngeal infections and not looked upon as primary. Two such cases have been reported by Lake.

The method by which the tubercle bacillus gains a foothold in the laryngeal mucosa probably varies in different cases. In a very few cases the organism may penetrate through the unbroken epithelium, as demonstrated by Wright. It may enter through the gland-ducts, and this is probably the line of infection when the lesions begins in the ventricles. The erosions caused by the traumatism of cough and the constant irritations of large quantities of decomposed sputum may become infected with tubercle bacilli. This method of infection is probably the most common way in ulceration of the true vocal cords. Lake has found in the epithelium minute abscesses, which he believes might finally have become tuberculous. Briggs calls attention to the early erosions as marking the stage where simple catarrhal laryngitis becomes tuberculous. Be the method of penetration what it may, the essential element in the infection is the enormous and constant dosage to which the larynx is subjected.

PAPER:

THE BANEFUL INFLUENCE OF PREGNANCY ON LARYNGEAL TUBERCULOSIS.

WOLFF FREUDENTHAL, M. D.

In bringing the above subject, which has been discussed so extensively abroad, to the attention of this large and experienced audience, it is not so much the desire of the writer to present his own observations, as to elicit a full discussion by all who have seen cases belonging to this category.

Right here permit me to mention one question, viz., that of primary tuberculosis of the larynx. While not denying the possibility[1] of a primary infection of the larynx, I shall eliminate it entirely from this paper solely to facilitate the discussion. Consequently we have to deal here with those cases of laryngeal tuberculosis which are associated with a pulmonary affection.

The first question that confronts us, is: Has pregnancy any influence in producing laryngeal tuberculosis in a person already afflicted with tuberculosis of the lungs? It is impossible to give a direct and satisfactory answer to that. Personally I believe that after the tuberculous virus has once gained entrance into the system (lungs) it is apt to form a new focus, wherever there is a locus minoris resistentiae. This law holds good for syphilis, carcinoma and other systemic diseases as well. Now if the larynx has been weakened previously by inflammatory attacks it will be more prone to become tuberculous at the slightest provocation. And gestation may well be considered such a factor. But a direct influence in causing laryngeal tuberculosis cannot be proven.

On the other hand, how is it, if laryngeal tuberculosis is already established? Has pregnancy then a deleterious effect? To this we must decidedly answer in the affirmative. Gestation can undoubtedly light up an old process that has practically been arrested,

1. I say purposely "the possibility," altho I have never seen a primary tuberculosis of the larynx. The cases shown by others as well as some doubtful ones in my own practice have not convinced me that the lungs were free from tuberculous invasion.

and it is a positive contributing cause in rendering worse an already existing one.

But let us consider for a moment certain physiological conditions belonging to pregnancy. With the growth of the foetus the mother requires not only nourishment for herself, but a constantly increasing supply for her offspring. Everyone is aware of the difficulty of nourishing any phthisical patient; how much more difficult is the task, if the maternal organism has to assimilate food for two instead of one being? Is it therefore, surprising that we so often fail in the attempt and that the pulmonary tuberculosis grows worse and with it, pari passu, the affection of the larynx?

Furthermore, with the growth of the child the abdomen expands and breathing becomes more difficult. Add to this some obstruction in the larynx, as for example perichondritis of the arytenoids, interarytenoid tumefactions, extensive infiltration of the vocal cords, etc., and you have a second factor that helps to impair both the general condition and with it that of the larynx as well.

During pregnancy there are certain other conditions that are not entirely physiological. They too have no beneficial influence upon tuberculosis, but on the contrary accelerate the process. Among these may be mentioned anemia, which here acts so perniciously, as well as a large variety of neuralgias.

A direct influence on laryngeal tuberculosis is exercised, however, by a symptom which is as painful, as it is deleterious, viz., vomiting. With each act of vomiting, which occurs more or less frequently in almost all women during the first months of pregnancy, there is marked irritation of any existing ulcerations in the larynx, with increase of the pain and dysphagia previously present. A similar, and more deleterious influence is exerted by the cough, whatever may be its source. It is easy to understand that under such circumstances the chances for a cure of any laryngeal affection are almost nil.

Occasionally paroxysms of coughing or vomiting are so severe as to induce premature labor. More often, if this does not occur, the patients get worse rapidly. If they survive the accouchement they die as a rule very quickly afterward. For though pregnancy is fraught with danger to the life of the phthisical mother, childbirth, according to all experience, is still more so. Few women with tuberculosis survive this period for any length of time, as the cardiac weakness consequent upon the loss of blood cannot be

repaired. This is the experience of many observers, though not of all. Now while there are differences of opinion in regard to the purely pulmonary cases, the views are somewhat more uniform concerning the influence of pregnancy upon tuberculosis, if complicated by involvement of the larynx. "Whether pre-existing or developing in the course of gestation, this form of tuberculous affection is always markedly increased during pregnancy. This typical unfavorable development of the disease and the danger of suffocation, in case tracheotomy cannot be performed in time, has led to the almost general acceptance of laryngeal tuberculosis as a positive indication for the artificial interruption of pregnancy." Thus speaks an obstetrician, H. Ehrenfest. (Peterson's Practice of Obstetrics, p. 346).

But do not let us dwell on the latter point as yet. First, we must answer the questions: What becomes of the mother, if we let gestation go on to the end of the normal period, and what becomes of the child? I have seen an unusually large number of such cases in my practice, in which the mother invariably died shortly after delivery, and the child in almost every instance. A gloomier outlook can, therefore, hardly be imagined, and the question of treatment was settled in my mind long ago. Great was my surprise, when, a few years ago, Professor A. Kuttner, of Berlin, took up the question and discussed it in that thorough manner we are accustomed to expect from him. Then it became evident that there was not such a unanimity of opinion as was believed before. K. gathered statistics from different sources and presents the following data: "* * * If we deduct from that number (100) three cases in which pregnancy was interrupted in the third or fourth month with beneficial result, only seven of the others survived the confinement, while all the rest succumbed quickly, following a deterioration of the laryngeal and pulmonary condition." These statistics furnish as bad a prognosis as my own. Nevertheless cases have been published, off and on, which were intended to show the fallacy of the rule first laid down by Kuttner. Thus Lennhoff exhibited a patient before the Berlin Laryngological Society who had survived childbirth and in whom the disease apparently did not run the usual fatal course. But at a later meeting of the same society Kuttner was able to show that even in this instance childbirth was the turning point for the worse. The woman ran

down rapidly after confinement and the outlook was in no wise favorable.

Soon after I started here in practice, the first of these cases came under my observation. The patient died three days after giving birth to a dead child. The next case occurred a year later with the same result, only that the child was kept alive with the greatest effort for two years. And so it went on. In every instance the mother died, and, with one exception, the child, too, until I finally established the rule that to let such a woman carry her child to the full term would mean the death of both.

In my history books there are ten cases, but I am positive that during the last twenty-one years I have seen many more, perhaps double that number. This may seem unusually large in the experience of one man, but it is probably due to my long connection with tuberculous hospitals in this city. It will not be necessary to give the histories of all in full, but three cases may suffice.

Case 1 (No. 2 in my statistics). Mrs. S., primipara, aet. 23, was seen by me in March, 1888. She was in the ninth month of pregnancy and suffered greatly from dysphagia. There were ulcerations on both vocal cords and the interarytenoid space. Both lungs were affected. Local treatment did not ameliorate the condition in the larynx, and at the normal end of gestation she gave birth to a dead child. Twelve days later she also died. There was no reason for inducing premature labor in this case, for it could not have saved the mother, as pregnancy was too far advanced.

Case 2 (6). Was seen in consultation with Dr. M. She was a sextipara and had acquired tuberculosis, apparently, before becoming pregnant. At least, cough had been present for some time. This was a wretched case. When I saw her first in October, 1885, she was in the fifth month and appeared quite weak. There was an infiltration of the right vocal cord, an ulcer on the anterior third of the left one, and the arytenoids showed oedema. Apparently only the right apex was tuberculous. For that reason I advised immediate resort to abortion, which was refused for religious reasons. Consequently the usual remedies were applied to the larynx, but the woman grew rapidly worse. When I was called in again five weeks later, the scene was almost tragic. To hear that wretched woman with her husky voice scold her family physician for not having treated her exactly as I had advised, and then begging his pardon, only to tell me a moment later that the whole medical pro-

fession did not know anything, and then again to pray of us to save her for the sake of her five little children, was pitiful, to say the least. One week later the Parcae cut the thread of her life.

Case 3 (8). A woman was brought at the close of her pregnancy to St. Mark's Hospital suffering from tuberculosis of one lung and of the larynx. In the latter there was a diffuse infiltration and a deep ulcer on the epiglottis. When I told the gentlemen present that, according to my experience, the prognosis *quoad vitam* was absolutely unfavorable, there was great surprise. On the sixteenth day after childbirth the woman died. The child, I hear, lived for several months. In this case, too, the interruption of pregnancy at such a late period would not have been of any benefit, but an early abortion might have saved the mother.

Such is the fate of the mother. How about the child? According to K., 72 to 73% of the children died immediately after birth or within a few weeks afterward. This is certainly a very low percentage, but still more perplexing are the statistics by Weinberg of Munich. Out of 321 children born alive, whose mothers died within a year, not less than 217 (67.9%) died in the first year. Of the 57 infants born alive, whose mothers died in the first twenty-eight days of the puerperium, as many as 37, or 78.8%, succumbed in the first year. And this apparently was the case with women afflicted only with pulmonary tuberculosis. In the presence of a complicating laryngeal tuberculosis, the prognosis for mother and child is so unfavorable that H. W. Freund[1] considers the former as the most important of all indications for the early interruption of pregnancy.

But there is another side to this question. Have we a moral right to sacrifice an unborn child in order to save a mother, whose life, as some maintain, is also doomed? This question has been asked me by physicians as well as laymen, and it is proper to answer it now.

In regard to the lives of children born of such mothers, the above statistics, which are supported by my own experience, speak a very clear and sad language. I recollect only one instance of a child that lived as long as two years. What became of it afterwards is unknown to me. As K. has shown, however, of those who survived for more than a year only a small percentage could be reared

1. Quoted from Gustav. Freitag's Inaug. Diss.: "Die kuenstliche Unterbrechung der Schwangerschaft wegen Tuberkulose." Breslau, 1906.

with the greatest care in well-to-do families, and even these children were weaklings.

What about the mothers? Is the percentage of deaths really equal 100%? Not in my experience, nor in that of others, when pregnancy is interrupted at an early stage. The reports on that point are meager, but every such case counts. Thus Levinger, of Munich, saw two cases. The first one which died in the puerperium, had a tuberculous tumor in the larynx that caused dyspnoea. Tracheotomy could not save the woman. In the second case, however, abortion was induced at the end of the fifth month, and the laryngeal tuberculosis was afterwards "permanently" cured, the lungs apparently, too.

The writer can report two cases that were saved.

Case 1 (No. 8 of my statistics). Mrs. F. M., aged about 30, mother of two very delicate children, was referred to me, on February 18, 1901, on account of pain in the left ear and persistent cough. The ear was normal, but there was an ulceration on the left ventricular band, and the upper lobe of the left lung was affected. Tubercle bacilli in the sputum. She was treated for several weeks and then left for Asheville, N. C. Afterwards she went to Germany, consulted Prof. v. Leube and other well-known men, and returned to this country at the end of 1902 greatly improved. I had warned her against conception, but upon the advice of a good friend she became pregnant, expecting in that way to get rid of all her trouble. Instead of this, the old laryngeal symptoms returned with renewed activity. When I saw her an immediate abortion was recommended, which was induced at the end of the third month of pregnancy by an obstetrician of this city. After that the larynx yielded to treatment again and was practically healed within six months. This lady is still living—it is now four years—although she had an attack of pleurisy two years ago. She is feeling so well that she has not consulted a physician for one year.

Case 2 (No. 10 of my statistics). Mrs. N. consulted me while in the fourth month of pregnancy. For several weeks she had been hoarse and felt a constant irritation in her throat. The aryepiglottic folds were swollen and injected, so were the ventricular bands. Right vocal cord serrated; t. b. c. of both apices; tubercle bacilli in the sputum. Immediate interruption of pregnancy was advised, and this was done within six days. Three weeks later the

patient went to the mountains, where she stayed a year and a half. Since October, 1903, i. e., more than three years, she has been back in New York City enjoying comfortable health.

These two cases demonstrate that a cure, or, let us say, a relative cure, does occur after the induction of a premature labor. These women have been restored to their families and are able to enjoy life once more.

Similar was the experience of de Bruine Ploos van Amstel, who, in a very elaborate paper on "Phthisis pulmonum and abortus provocatus" (*Beitraege z. Klinik der Tub.*, Bd. 7, 1907), reaches the conclusion that the less far advanced tuberculosis is in a gravida the more urgent is the indication to interrupt pregnancy in the interest of the mother; in that way the possibility of saving a relatively healthy woman is the greatest.

Before laying down a general rule, however, in regard to the artificial interruption of pregnancy, I asked several prominent obstetricians of this city for their opinion. It is remarkable to note how very few cases of laryngeal tuberculosis had been observed by these gentlemen in spite of their enormous experience in obstetrical work.

Thus, Dr. J. W. Markoe, in an experience of sixteen years at the Lying-In Hospital, during which time 49,000 women had been confined, does not recollect one such instance. The fact is that such cases consult the laryngologist rather than the obstetrician, and, furthermore, they are not admitted to hospitals.

Dr. J. Clifton Edgar remembers only one case that lived a short time after delivery at the fortieth week.

Dr. S. Marx reports the following cases:

Case 1. A well-known singer, pregnant in the third month, showed incipient pulmonary tuberculosis and "ulcerations in the larynx." Abortion was induced and the patient afterwards sent away. She is living now in this city in full possession of her health and voice. The abortion occurred six years ago.

Case 2. Young woman, very similarly affected as the former, died within six weeks after confinement at the regular time. Dr. Marx makes it a practice to induce abortion as early as he can.

Dr. J. B. Cragin writes: "In reply * * * would say that in the last 8,000 deliveries at the Sloane Hospital we have had 13 cases of tuberculosis, but in none of these was there any special laryngeal involvement. This may be accounted for by the fact that

we do not usually take into the hospital cases with active tubercular inflammation. I remember delivering only one woman with marked laryngeal tuberculosis, and she died within a few months following her confinement."

All these colleagues, and a few more consulted by me, advise immediate interruption of pregnancy, if the case be seen early. If seen late, they generally wait in the interest of the child.

It seems to be the general opinion that the tubercular process is greatly stimulated by gestation and labor. Only Austin Flint, Jr., has seen a few cases improve during pregnancy "by ordinary anti-tubercular treatment." This we must consider as a decided exception, as it is not in accord with what others have observed. Still such cases ought to be recorded in full, if the above question is ever to be settled definitely.

In going over the literature, we come across several other cases improved during pregnancy and after delivery, even without any treatment whatsoever. It must be repeated, however, that in most of these it has not been proven satisfactorily that the laryngeal affection really was tuberculous. A slight swelling here or there or a redness in a consumptive patient, while certainly to be treated with every care, is no positive symptom of laryngeal tuberculosis, and for that reason should not be considered an indication for the interruption of pregnancy. All of my cases were, unfortunately, of such advanced type that the diagnosis could not be doubted for a moment.

But is there nothing that can be done for such patients excepting the artificial interruption of pregnancy? The usual remedies that leave us in the lurch so often are even less reliable here. The disease makes rapid progress, and the only thing left to do in some instances is tracheotomy as an indicatio vitalis. Kuttner seems to think favorably of the latter, even as a means of cure in some cases. I am not so optimistic, but would rather reserve tracheotomy for such patients who present marked dyspnoea. Then it will be a life-saving means for the time and no more.

In all other cases, however, especially those showing a diffuse tubercular affection of the larynx, we have to fall back to an early interruption of pregnancy as the only means of saving the life of the mother.

There are some exceptions to this rule. If a woman is first seen at the end of pregnancy, then a few weeks make no difference, and

it is best to wait in the interest of the child. Otherwise the life of the child can be regarded as a negligible factor, since it is lost, anyway, in almost every instance.

Furthermore, if the progress of the disease is so rapid that we are positive the mother will die shortly, then, too, it is useless to induce premature labor. But these cases should be weighed very carefully, since nothing is so surprising as irregularities in the course of tuberculosis. Patients sometimes recover in a marvelous way, while others succumb as unexpectedly.

The medico-legal side of this question has been ably discussed by K. and several prominent jurists abroad, but I do not feel inclined to enter into it here. It seems hardly possible that any judge in this or any other country would condemn a physician because he acted in the purest interest of his patient, of humanity, and often even to his own detriment, when he advocated an early interruption of pregnancy as the only possible salvation for the mother.

For that reason, it is so much the more our duty to be extremely careful in making a diagnosis and in advising our patients what to do in such a dilemma.

PAPER:

A CERTAIN PHASE IN THE DIFFERENTIAL DIAGNOSIS OF TUBERCULOSIS AND SYPHILIS OF THE UPPER RESPIRATORY TRACT.

LEE MAIDMENT HURD, M. D.

It is not my purpose to differentiate in parallel columns syphilis and tuberculosis as they appear in the upper air passages, for it would take too long and be detail ad nauseam before such an assemblage. I only wish to call your attention to certain conditions which are confusing to both the microscopist and the clinician:—Inflammations and ulcerations which may clinically closely resemble either syphilis or tuberculosis, or perhaps neither. The correct diagnosis in these cases is of much importance for the patient's peace

of mind and future welfare. More than likely some of the reported cases of cured laryngeal tuberculosis have been syphilitic.

This short article was prompted by the following three cases:

Mary K., aet. 25. Servant. Native of Finland.

Noticed nasal obstruction about one year ago, which gradually grew worse. There was elicited the fact that there had been some pain in naso-pharynx and across bridge of nose before she noticed the nasal obstruction. Other than this there were no local or general symptoms, and to all appearances she is in the best of health.

There is no family history of tuberculosis, or any tubercular people in the family in which she is employed. On examination the nose and pharynx were found normal, but on examining the naso-pharynx a growth occupying the vault and posterior wall was found with a slightly nodular surface covered with mucous membrane of normal color and appearance. I removed the growth with forceps and submitted it to Dr. J. Wright, who reported as follows: Microscopic examination of a piece removed by forceps shows that in mounting, the hardened specimen crumbled from the hardening process. Many of the fragments show very plainly perfectly typical tubercle. Centres of tubercle granulum or necrobiosis are surrounded by a rim of new connective tissue in attempts at repair. In a few of these areas may be seen a typical Langhan's giant cell. Some of the round cell tissue apparently at a distance from the tubercles is altered somewhat by chronic inflammation and contains no lymph nodes. The tuberculous tissue, or rather the tubercle granula, are not immediately under the epithelium which is fairly normal columnar, but separated from it by a rim of lymph cells. This is the second case only which I have seen in which there were multiple tubercles in a posterior lymphoid hypertrophy, the other having been from the service of Dr. Chappel and reported by me with a color plate in the N. Y. Med. Journal, Sept. 26, 1896. Perhaps, owing to the scantiness of the lymphoid tissue there being no nodes, it is not accurate to call this tubercle of the adenoid, but of the naso-pharynx.

A search of 10 sections failed to reveal any tubercle bacilli.

March 15.

After receiving this report, I removed a small remaining piece near the posterior choanae, and two guinea pigs were inoculated intra-peritoneally. Both of the pigs lived, and one was killed this week, two months after the injection, with the following findings:

June 6th, 1906.

One of the guinea pigs inoculated two months before with pieces of the growth removed from the naso-pharynx beneath skin of abdomen, showed enlarged inguinal glands. One of these on section showed fairly typical tubercle. The other guinea pig showed beginning round celled infiltration (presumably tuberculous) of the smaller bronchi.—J. WRIGHT.

Although latent tuberculosis of the lymphoid tissue in the vault is not an infrequent occurrence, a purely tubercular growth like this I believe to be unique.

G. S., June, 1906. U. S. Aet. 57. Brass worker. Married.

Family history is clear. Previous history, usual children's diseases. Denies both gonorrheal and syphilitic infection, although he says he has lead a wild life, and could very well have been infected. Seven months ago gradually became hoarse without cough or other symptoms. He states he has had previous attacks of hoarseness which have disappeared of their own accord. The lungs are normal. Repeated examinations of sputa fail to show tubercle bacilli. On examining the larynx we find ulcerations along the posterior 2-3 of right vocal cord and post. 1-3 of left cord. The laryngeal mucous membrane is slightly congested. The ulcerated area is covered with a white exudate, and the ulcerations are not very deep; the edges do not seem undermined.

A section was removed and sent to the Pathological Laboratory, Dr. Jonathan Wright reported as follows: "The specimens on section show areas of coagulation necrosis, and in them or near them typical Langhan's giant cells and epithelioid cells. Subsequent sections examined for tubercle bacilli; none found." Dr. Harlow Brooks, who saw the same sections, reported as follows: "The coagulation necrosis is rather less definite than is usually the case in tuberculosis. There is general hyperplasia of the connective tissue with thickened arterial walls, with periarteritis most marked. Inclines me to think it is syphilis."

This case was injected with salicylate of mercury, gr. 1, weekly, for nearly six months. Potassium iodide was prescribed, but I doubt if it was taken regularly. For the last six months the ulcerations have been entirely healed, which shows beyond doubt that it was syphilitic. He was under mixed treatment several months before the ulceration showed any tendency to heal.

L. W. Aet. 45. Male.

Gives neg. family history. Previous history of syhpilis twenty years years ago. Is an alcoholic. Several years ago while drinking to excess he had a cough with slight night sweats. He has had no cough or night sweats for three years. Four months ago he became gradually aphonic. Two months ago dyspnoea developed, which has steadily increased until at present he labors considerably for air. On examiantion, the lungs and heart were normal. No syphilitic scars in nose or throat. His larynx contains so much thickened and inflamed tissue that the merest chink remains for breathing. The false and true cords are greatly thickened, with ulcerations on the true cords; also considerable subglottic infiltration below left cord with an ulcerated surface. The ulcerations did not appear deep, or to have undermined edges.

The laryngeal mucous membrane was deeply congested with some mucous secretion. I made a clinical diagnosis of syphilitic infiltration with ulceration. Dr. Wright was inclined to the same belief, but thought it might be malignant. The patient was admitted to the Manhattan Eye, Ear and Throat Hospital, and placed upon mercurial injections and Pot. Iod. in large doses. This was changed at the end of a week at the suggestion of Dr. Wright to Bichlor. and K. I. by mouth. The third day after admission a tracheotomy had to be performed. The mixed treatment was continued for five weeks without apparent effect upon the laryngeal condition, except that dyspnoea decreased somewhat. He wore the tracheotomy tube most of the time and the rest that this gave the larynx may account for the improved breathing. The secretion from the tracheotomy tube was repeatedly examined for tubercle bacilli without results, and a portion of the growth was removed from over the left arytenoid and examined by Dr. Wright, who reported as follows: "The epithelium is flat, undergoing great hyperplasia and dipping down with the stroma in all directions, there being in these sections some separate islands of it. It is sharply differentiated from the stroma and has no appearance of malignancy. In the stroma are large areas of necrosis of tissue, containing giant cells of Langhan's. So far as these appearances go, we are compelled to assume the presence of tuberculosis, though there are some thickened vessel walls which remind one of syphilis."

Dr. Harlow Brooks, who has examined the same slides, states that microscopically, it looks like tuberculosis.

After remaining five weeks with us, he was transferred to the City Hospital, with a note from Dr. Wright stating that he was inclined to believe the case tubercular. At the City Hospital his sputa was repeatedly examined without results, and he was discharged at the end of three weeks when he again reported to the clinic. I found his laryngeal condition considerably improved; the thickened tissue had gone down about 50%. He had received no medication while in the City Hospital. He then went on a spree, and I have not seen him since.

I attribute his improvement to the mixed treatment that he had before going to the island. His tracheotomy tube was corked all the time that he was away, so we cannot say that the improvement was due to rest of the parts. The improvement in this case was most marked, and leaves little doubt in my mind that if treatment had been continued long enough the laryngeal condition would have been completely cured.

Dr. Eugene Hodenpyl, who examined the slides from these cases, January 2, 1907, reports as follows:

"I have examined microscopic slides of your cases: To be brief, each case, I think, presents in a fairly typical fashion, the morphological lesion."

Certain phases of syphilis of the mucosa are histologically identical with tuberculosis. I refer to those in which there may be easily demonstrated under the microscope a number of giant cells. These giant cells do not seem to differ in any way from the so-called Langhan's giant cells.

Given an area of chronic inflammatory action of the mucosa, crowded more or less with round cells, whether there is any surface loss of epithelium and consequent ulceration, or not, the pathologist must be careful in making an unqualified histological diagnosis of tuberculosis, because in every field he sees one or several giant cells.

The presumptive diagnosis, whatever the clinical history, should be syphilis when the marked vascular thickening and the number of giant cells are out of all proportion to and more prominent than the coagulation necrosis, unless the tubercle bacilli can be demonstrated in the tissues.

It is true that giant cells are much more often found in tubercular than in syphilitic foci of chronic inflammation, but when they do

occur in syphilis they are much more numerous than is usual in tubercular cases.

I refer especially to swellings and ulcerations of the mucosa, granulomata, not large gummata or extensive tuberculous softening like lymph nodes,—tertiary syphilitic processes of the upper air tract are usually destructive, often invading the bone; the granulomatous infiltrations of the mucosa are of less frequent occurrence.

Manasse (*Virchow's Archiv*, 147, 1897,) states that the presence of giant cells or of ordinary nodules containing the giant cells is not positively available in the determination of the question as to spyhilis or tuberculosis, these features being sometimes present, sometimes absent in either disease. It is hardly necessary to mention that caseation and other degenerative processes occur in syphilis as well as in tuberculosis, and that the presence or absence of these retrogressive metamorphoses does not constitute a distinctive sign between these two diseases; neither is the production of connective tissue which leaves familiar scar formation, a reliable sign, as it is also found in fibrous tubercles, etc.

Arteritis, leading to vascular obliteration, is often quoted as a characteristic feature of syphilis, and as a matter of fact, this condition is almost constantly found. However, this vascular change is not entirely conclusive, for it is met with in tubercular conditions as well.

The giant cells perfectly resemble Langhan's giant cells met with in tuberculosis. Certain authors claim that the giant cells originate by individual growths from a single cell; others, that several cells are concerned in their growth. The general tendency seems to be to regard them as cross sections of pre-existing channels, either vascular, lymph, or glandular, on account of their parietal nuclei. But whatever their true origin, it may be assumed as a foregone conclusion, that it is the same in both tuberculosis and syphilis.

The only conclusion that can be drawn is that the microscope and the clinical picture cannot be unqualifiedly relied upon; in fact, unless the tubercle bacilli can be demonstrated in the tissues or by inoculation, we should consider the condition syphilitic; and also that the ordinary doses of mercury and potassium iodide will show small results in these connective tissue conditions. It requires mercury and potassium to the limit, administered over a considerable period of time, to show satisfactory results.

DISCUSSION OF TUBERCULOSIS SYMPOSIUM.

Dr. Clement F. Theisen, of Albany, N. Y., said that in considering the differential diagnosis between laryngeal tuberculosis and laryngeal syphilis the hypertrophic or hyperplastic form of the former should be borne in mind. In this form of tuberculosis distinct hyperplasia of the tissues existed, which might go on through the entire course of the disease without ulceration. Ulceration, however, might result in the later stages of the disease. The syphilitic tumor, the granuloma, usually developed around an ulcerative process, which as a rule appeared first. A distinct tuberculosis tumor sometimes occurred in the larynx and did not ulcerate.

Dr. George T. Ross, of Montreal, Canada, asked Dr. Levy what percentage of primary laryngeal lesions existed in his cases exclusive of constitutional infection. Dr. Wood's case referring to infection through the tonsil was interesting. He asked if any examination was made with reference to other pathological germs besides the tubercle bacillus known to exist in the so-called healthy mouth, such as the diplococcus pneumoniae, and others capable of producing infective symptoms. He agreed with Dr. Freudenthal that tuberculosis in pregnant women meant very often a bad prognosis for her as far as life is concerned; for, although an apparent quiescence existed during the progress of gestation, after the accouchement renewed activity ensued in the disease and this often hastened the end.

Dr. James. F. McCaw, of Watertown, N. Y., cited a case, still under observation, in which a presumptive diagnosis of tuberculosis had been made. The patient was a girl seven years of age, who, shortly after playing on a dump-heap in the town to which her family had recently moved, first noticed a small ulceration on the side of the nose. The process spread fairly rapidly and involved almost to the angle of the eye and well down on the cartilaginous portion of the same side. The cervical lymph nodes, well down to the clavicle, anterior and posterior chains, later became involved and were subsequently removed. It was thought that a general infection had taken place. This was evidently a case in which general infection took place from a local lesion on the external surface of the nose.

Dr. J. Price Brown, of Toronto, Canada, cited a case referred to him as tuberculosis of the larynx. The patient, a young man, gave a family history of tuberculosis. The vocal cords were markedly thickened and there was considerable infiltration. No tubercle bacilli were found upon examination. The patient was put on antisyphilitic treatment and had greatly improved. The voice, which was practically lost, had returned.

Dr. Robert Levy, of Denver, Colo., said the question raised by Dr. Wood called attention to the work done by Walsham, who concluded from the many autopsies performed by him that tuber-

culosis can often be traced through the tonsils, involving the lymphatic glands in the neck, and later the bronchial glands and lungs. Answering Dr. Ross' question, he said in no case had he been satisfied that the infection in the larynx was primary. He had seen a great many cases in which the only manifest lesion was in some portion of the upper air passages, but in no instance would he conclude that this was a primary lesion, unless the patient should die of some intercurrent disease and autopsy reveal beyond a doubt that there was no other manifestation of tuberculosis.

Dr. Wood, in closing the discussion, said that he had seldom been able to demonstrate tubercle bacilli in tonsillar tissue and that he did not consider that the demonstration of their presence was necessary to a diagnosis of tuberculosis when the typical histologic changes were present. He considered that the recent work of Beitzke was probably the best and most thorough that had been done with relation to the possibility of infection of the pleura or bronchial lymph nodes directly from the cervical lymph nodes.

Dr Freudenthal, in closing the discussion, said the question of primary tuberculosis of the larynx had occupied his mind for a long time, and he was not convinced that he had ever seen a case. A case had recently been demonstrated at the New York Academy of Medicine as primary tuberculosis or *lupus* of the larynx. He questioned the diagnosis at the time, and shortly thereafter it was demonstrated that the patient had tuberculosis of the lungs. He had found hypertrophy of the lingual tonsil in a great many cases of tuberculosis of the larynx.

PAPER:

A CASE OF PRIMARY CARCINOMA OF THE UVULA. OPERATION. NO RECURRENCE.

CLEMENT F. THEISEN, M.D.

Primary carcinoma of the uvula is rare, and a study of the literature of recent years revealed the records of only a few cases in which the malignant process was confined absolutely to the uvula with every other part of the throat showing normal conditions.

The case I wish to report presents a number of interesting features.

Mr. P. G., aged 52 years, consulted me on March 17, 1906. He stated that for the past three months he had felt a growth in his throat, and at times had had slight pain radiating to the ears. His family history is an excellent one.

On examining the throat it was found that the uvula had been transformed into a somewhat nodular growth. The tumor was firm to the touch and so large that it disturbed him a good deal while eating. His voice was not affected particularly, nor did the growth interfere with respiration. Just at the junction of the

Primary Carcinoma Uvula. (Case of P. G., aged fifty years.)

uvula and soft palate the mucous membrane appeared normal. The growth had not quite extended to the soft palate, nor was there any induration of the soft palate or any of the surrounding tissues. The rest of the pharynx was absolutely normal, and the naso-pharynx, larynx and nose, also presented normal conditions. A portion of the anterior surface of the growth was smooth, but on the sides and at the tip of the growth, there were firm, nodular excrescences. No enlarged glands could be felt anywhere in the neck. The growth was removed including a small portion of the soft palate on March 20, 1906. The wound was cauterized with a solution of chromic acid. There was very little hemorrhage during the operation.

It is now over fourteen months since the operation and there is absolutely not the slightest indication of a recurrence, the

pharynx presenting a perfectly normal appearance. It is in fact, rather difficult to find the scar of the operation.

The patient who before the operation was in rather poor condition, has gained over thirty pounds in weight and is perfectly well. I saw him only a few days ago.

The specimen was sent to the laboratory for examination and the report is as follows:

BENDER HYGIENIC LABORATORY.

Case of Dr. Thisen. No. 06-392.

Specimen consists of a pinkish white nodular mass measuring with pedicle 3.5x2.5 cm. The main body of the tumor is a smooth globular mass about 1.3 cm. in diameter and from this protrude two coarsely lobulated papillary masses; the larger measures 1 cm., the smaller .5 cm., in diameter. The body of the tumor is firm and resistant but not as hard as the papillary masses. The pedicle, which measures 1 cm. in length and .6 cm. in width, is soft, greyish white on section and covered by mucosa except at point of excision.

Microscopic Description—Sections through the main tumor mass show large oval or irregular masses of epithelial cells, lying in alveoli formed by fairly definite strands of fibrous tissue. These epithelial cells are very closely arranged, uniformly oval or cuboidal, and do not resemble the ordinary stratified epithelium. Here and there through the larger foci, are a few areas of degeneration and slight leucocytic infiltration. The connective tissue stroma is poor in cells. The capsule of the tumor is formed by a thin layer of squamous epithelium, beneath which is a slight infiltration of lymphoid cells.

Sections through the larger papillary mass show in a general way a similar structure but with a larger amount of fibrous stroma. The squamous epithelium covering this portion shows a tendency to papillary growth. Here is seen also a direct transition of cells from the surface epithelium to the deeper alveolar masses.

Sections taken through the pedicle of the tumor close to the line of excision show a moderate hyperplasia of the surface epithelium but no infiltration of sub-epithelial tissues.

MICROSCOPIC DIAGNOSIS—CARCINOMA OF UVULA.

DR. ROBERTSON.

I know of course that there may be a recurrence, but the statement that Kyle makes, that after the removal of carcinomata of the

uvula or soft palate there is an early recurrence, is not borne out by some of the reported cases.

Two very interesting cases of epithelioma of the uvula and soft palate have been reported by Downie and Browne.

The patient in Downie's case was a man aged 56 years. He had had a sore throat for several months, more marked when he swallowed. The uvula was greatly enlarged and the greater part of its surface was ulcerated. The mass was firm and bled easily. There was no involvement of the regional lymphatics nor cervical glands. The growth was removed and there was no recurrence for seventeen months.

Browne had reported two cases. In one of the cases, a man aged 48 years, the uvula had been transformed into a warty, thickened mass, and the soft palate was also involved. There were no enlarged glands in the neck. The growth was removed with scissors. A rapidly growing recurrence occurred in the cervical glands. Patient lived 19 months after the operation.

In his second case, a man 52 years old, there was a warty growth at the free border of the left anterior faucial arch, slightly hanging over the supra-tonsillar fossa. A piece removed for examination confirmed the diagnosis. No operation was performed.

Friedmann observed a case in Prof. Baginsky's polyclinic. A man, aged 49 years, complained of some pain in swallowing. The uvula had been changed into a yellowish red tumor the size of a cherry. There were no regionary metastases. A piece removed for examination confirmed the diagnosis of carcinoma.

Friedmann states that up to 1905, he had only been able to find reports of 4 cases of primary carcinoma of the uvula.

Harmon Smith has reported a case occurring in a man aged 51 years. There was a mass on the uvula as large as a strawberry, the induration extending to the soft palate. There was no involvement of the glands on the right side but there was a large gland in the left sub-maxillary region. The microscopical diagnosis was epithelioma. The growth, including part of the soft palate was removed. A month afterward there was a beginning recurrence in the wound. Another operation, which included the large gland in the neck, was performed, and five months later, after this operation there had been no recurrence.

In Raynors' case, a man aged 66 years of age, the uvula was elongated and oedematous, and behind it there was a nodular mass attached to its posterior surface and extending ½ inch below the tip. The growth was removed with the cold snare. The pathologist reported that the growth was atypical, but in all probability a carcinoma. There was a recurrence in the wound a little over 5 months after the operation.

Primary Carcinoma Uvula. (One-half actual size.)

Oppenheimer has reported a case of primary epithelioma of the uvula in a man aged 81 years, in which no operation was performed, and McCaw has reported a case in which the soft palate was also involved. His patient, a woman aged 37 years, was apparently cured after a partial removal of the growth followed by X-Ray treatment.

Lieras has reported a case of epithelioma of the uvula and faucial pillars from Moures' clinic. There was no glandular involvement in his case.

In Zurakowski's case, a man 71 years of age, the uvula was thickened and firm to the touch. There was ulceration of the upper part of the uvula and part of the soft palate. The submaxillary glands were enlarged. The microscopical diagnosis was carcinoma keratodes.

Newman has reported a case of adeno-carcinoma of the left tonsil, soft palate and uvula.

It was removed through the mouth. No recurrence for six years.

Of the cases mentioned in the writer's paper (the cases reported in the literature of recent years), in only six, was the malignant growth confined to the uvula.

They are Downie's, Friedmann's, Smith's, Raynor's, Oppenheimer's and the writer's.

In Downie's case there was no recurrence for 17 months after operation.

Friedmann's—No operation..

Smith's—Recurrence three months after first operation, no recurrence five months after second operation.

Raynor's—Recurrence five months after operation.

Oppenheimer's—No operation.

Theisen's—No recurrence 14 months after operation.

The fact that in some cases of malignant growths of the uvula and soft palate there is no recurrence for such a long time after operation (Newman's case six years), would go to show, that such growths involving the muciparous glands are of slow growth, with less chance for recurrence than when they extend through the lymph channels, as would be the case with malignant growths of the tonsil.

Since writing this paper the author has seen Dr. Harmon Smith's case. It is now about three years since the last operation, and there was no recurrence. The pharynx appears perfectly normal.

BIBLIOGRAPHY.

1. Oppenheimer, Seymour. Primary Epithelioma of the Uvula. Trans. Amer. Laryngol. Rhinol. and Otol. Soc., 1901.
2. McCaw, J. F. *Ibid.*
3. Downie, Walker. *Scottish Med. and Surg. Journ.*, Vol. IV, p. 36, 1899.
4. Zurakowski, W. Ein Fall von primarem Krebs der Uvula. *Virchow's Jahresber*, Vol. 11, p. 333, 1896.
5. Browne, Lennox. *Scottish Med. and Surg. Journ.*, Vol. IV, p. 220, 1899.
6. Smith, Harmon. *N. Y. Med. Journ.*, Apr. 29, 1905.
7. Raynor, F. C. A case of malignant growth of the uvula. *The Laryngoscope*, Feb., 1902.
8. Newman. A Case of Adeno-carcinoma of the Left Tonsil, Soft Palate and Uvula. Trans. London Laryngol. Ass., Jan. 8, 1896.
9. Lieras. Epithelioma de la luette et du voile du palais. Societe d' Anatomie et de physiologie de Bordeaux. No. 1, Jan. 1897.
10. Friedmann. Ein Fall von Carcinom der Uvula. *Berl. klin. Wchnschr.*, No. 15, S. 444, 1905.
11. Kyle, D. Braden. Diseases of the Nose and Throat, p. 231, 1899.

DISCUSSION.

Dr George B. Wood, of Philadelphia, said that he had had two cases of malignant tumor of the tonsil during the past winter. One patient had a bad recurrence, but the other was doing fairly well up to date. Both of these cases were sarcoma, one belonging to the round-cell and the other to the spindle-cell variety.

PAPER:

A SECOND REPORT ON A CASE OF EPITHELIOMA OF UVULA, OPERATED FIVE YEARS AGO, WITHOUT RECURRENCE.

JAMES F. McCAW, M. D.

This case of primary epithelioma of the uvula and soft palate was seen in consultation on November 19th, 1901, through the courtesy of Dr. E. S. Willard, of Watertown, N. Y., a detailed report of which will be found in the transactions of this society for 1902. I will here briefly rehearse the important points in the case that we may better understand the nature and extent of involvement when first seen.

The history of obscure abdominal growths in two sisters, one of whom died from an attempt to remove the growth several years before. The patient, a female, had always enjoyed good health, and with no other throat difficulty, except an occasional attack of quinsy. Her present trouble began about eight months before coming under observation with soreness and slight throat irritation. In a short time several small ulcerations were noticed on the soft palate. Under treatment for "cancred sore throat" these seemed to improve, but never entirely disappeared. About October 1st, 1901, the soft palate began to enlarge and very rapidly fill the throat. This was quickly followed by dysphagia, pain, muffled intonation and soreness in the cervical muscles. Six weeks later, when first seen by the writer, a mass involving the uvula, soft palate, posterior faucial pillars the right lateral and a portion of the posterior pharyngeal wall was seen. The mass almost filled the right side of the pharynx, presented an irregular nodulated outline ulcerated and necrotic surface and a dense cartilaginous feel to the touch.

The treatment employed was thorough excision of the growth with electro-cautery knife curetting and deep cauterization of the raw surfaces, and subsequent irradiation with the Roentgen Ray. After two operations, according to the above method, the last,

March 7, 1902, followed by exposing the areas to the influence of the X-ray, the parts healed kindly after about twelve weeks of such treatment.

This case has been kept under constant observation and regularly examined at intervals of about six months ever since, either by her family physician, or myself, and at no time has there been the slightest indication of a recurrences. From a weak, frail, anaemic and despondent individual, she has gradually developed into a hardy, robust and cheerful person, gained thirty pounds in weight, and is in better physical condition than for years. The palatal and pharyngeal muscles functionate with perfect ease, the scar tissue scarcely noticeable. The only apparent deformity being the loss of the uvula, and slight tention of the velum palati; the posterior faucial pillars forming a narrower arch.

In view of the fact that this patient has remained free from the slightest indication of a recurrence for a period of five years, warrants us in assuming that the original process has been adequately removed.

In this connection the question arises in my mind; may we not have different degrees of malignancy inherent in growths, pathologically of the same type? The same, for example, as we have different degrees of severity in cases of diphtheria, typhoid fever, follicular tonsillitis, and a host of other conditions. This, together with the variable resisting power of different individuals would theoretically account for the cases of reported cures of malignant disease. The whole question of malignancy? where it begins; where it ends, and its aetiology is still so indefinite that we are unable to say the length of time that must elapse before a recurrence or metastases are improbable. Until we have more exact knowledge of these important factors, we think it wise not to report our cases of malignant disease as *cured*, but as having remained so long without a recurrence or metastasis. My case today, after five years, is still without a local recurrence or discoverable metastatic deposit.

PAPER:

OBSERVATIONS ON THE TEMPORAL BONE.

R. JOHNSON HELD, M.D.

The study of the surgical anatomy of the temporal bone is always interesting. There has appeared from time to time much literature on this subject, with many recorded observations, notwithstanding which there still exists considerable diversity of opinion not only as to the exact status of some of the more important points in the anatomy of this bone, but also as to the surgery of the mastoid process.

The constancy of certain familiar landmarks, the size and position of the several parts of the temporal bone and their relation to neighboring organs of importance are to my mind of sufficient interest to warrant the report of my findings from the examination of material upon which the work of this paper is based.

I. MATERIAL.

The material upon which the observations quoted in this paper are based consisted in the examination on the cadaver of 463 adult temporal bones. Of this number 249 were males and 214 females. In age they ranged from 19 to 79 years.

II. VARIATION OF CONTOUR.

The study of the comparison of the fully developed mastoid process in different adult individuals presents such variation in contour and structure as to make it impossible to establish a definite description as to the exact exterior and interior of a mastoid process. There exists no bone in the human body which presents so many deviations, both externally and internally, as does the normal mastoid process of the temporal bone.

Outwardly the mastoid process may be flattened or round in contour. The tip may be vertical, oblique or curved. The outer surface may be smooth and hard, or it may present irregularities and numerous openings for the passage of blood vessels. The cortex may be exceedingly thin, not more than .5 mm. in thickness, or

it may be as thick as 10 mm. Certain limited areas may be thin and the surrounding areas thick. The same multiplicity of variations as noted in the examination of the exterior is found in the examination of the interior.

Apart from the variations due to the development of the bone from infancy to adolescence, certain other conditions no doubt influence the character of the contour of the mastoid process and its interior, and while it cannot be said with any degree of certainty as to what will be the character of a mastoid process from its external appearance, yet it can with reasonable assurance be assumed that the mastoid process of a well developed and muscular man will be of large size, with prominent surface markings, and a spacious interior.

The dolichocephalic skull will present a larger mastoid process than a brachycephalic skull, a male a larger process than a female, and likewise a muscular man a larger and better developed mastoid process than a non-muscular man.

Since, therefore, we have large broad processes and small narrow ones, we are justified to suppose that when the process is large that the essential structures contained within it will be separated from each other much more than when the process is small, when the structures must necessarily be in closer apposition in the more confined space.

The arrangement of the cellular tissue of the mastoid process is also subject to vagaries of structure.

III. SYMMETRY.

The symmetry of the two mastoid processes in an individual is at times important from a surgical point of view. In the examination of 119 crania for determining asymmetry of these processes, I found the mastoids decidedly asymmetrical 9 times, or 7.5% of cases examined. Blake,[1] in an examination of 300 crania with reference to asymmetry found that 10, or 3 1-3%, had markedly asymmetric mastoid processes.

In a general way, it may be said that the mastoid process is conoidal in form, with a direction downward and slightly forward. Contrary to the claims made in most text-books that the anterior wall is distinctly vertical, the anterior wall may be vertical or may incline obliquely forward, or the tip may be curved in a forward direction. My observations showed the anterior wall vertical in

69% of cases examined, inclined obliquely forward in 26%, and curved forward in 5%.

The posterior border inclines downward and forward at an angle of about 45 degrees.

IV. SURGICAL LANDMARKS.

On the external surface of the mastoid process are noticed several landmarks of surgical importance, the suprameatal spine or spine of Henle, the suprameatal or supramastoid fossa, the remains of the squamo-mastoid suture and the mastoid foramen.

Of the three portions of the temporal bone, the squamous and petrous portions enter into the formation of the mastoid process; the squamous above and in front, and the petrous behind and below. The junction of these two parts form the squamo-mastoid suture. This suture situated on the outer aspect of the mastoid process runs from the parietal notch downward and forward to a point behind and below the external auditory meatus, external to the styloid process.

I have found it present as an open suture 9 times, and the remains of the suture evidenced 74 times, or 16%.

This squamo-mastoid suture belongs more properly to the infant bone, though frequently it persists until puberty and in some cases remaining permanently unossified. In adult life usually some traces of it remain and it is recognized during operation by the firm adherence to it of the overlying periosteum, which, when elevated, leaves a white line of shreds upon the surface of the bone. MacEwen[7] reports finding the suture open, in adults, 6 times during operation. (I observed this condition twice on adults, during operation.)

The suprameatal spine of Henle from the reliance placed upon it by many otologists as an infallible guide when operating, makes it one of the most important landmarks when present.

The spine varying considerably in size and prominence has nearly always the same relative position. It is a more or less prominent projection of bone situated above and behind the posterior-superior quadrant of the external auditory meatus, curved concentrically to the circumference of the meatus, with its upper portion slightly anterior to the lower portion. In size, it may be large or small. It may project prominently, or it may be flattened in appearance, or it may be entirely absent, or so slightly developed as to be overlooked, and can only be recognized by an experienced eye or felt

by the finger. Usually its development is in ratio to the development of the balance of the surface markings of the temporal bone.

It is not constant. This differs from the views formerly held by most otologists, but at present there still exists differences of opinion as to just how often its absence may be noted. Yearsley[3] in the examination of 2,000 temporal bones found the spine absent 160 times, or 8%. In addition, he found the spine very poorly marked in 12.57%, being recognized only by the trained eye and finger. Broca[4] states that Kiesselbach noted its absence 18 times in 100 temporal bones which he examined; Schultze, 11 times in 120, and Lenoir, once in 200. My results of the examination of 463 temporal bones gives it absent 56 times, or 12%. It was barely demonstrable in 41 other specimens.

When present, the suprameatal spine is a valuable landmark, and can be depended upon to furnish an excellent guide for entering the mastoid antrum, but, since the observations given above show its absence 246 times in 2,883 cases, or 8.5%, too much stress must not be placed upon this as a landmark when operating.

The relation of the spine to the floor of the middle cerebral fossa showed the average distance between its upper part and the floor to be 6 mm. In one instance the floor was as low as the spine. Randall,[5] in the examination of 1,000 skulls, found it as low as the spine in but 5 cases.

Lying immediately behind the suprameatal spine is a depression called the suprameatal fossa. This landmark is more constant than the spine, therefore more useful as a guide. It represents the adult condition of the spongy spot seen in the infant, up to the second year of life. It is the external boundary of the antrum. In the infant its position varies with the position of the antrum, but in the adult its position is constant.

It may be a large excavated depression triangular or crescentric in shape, or it may be a mere chink, or slit or it presents as several large vascular foramina or many small ones. Yearsley[3] reports the finding of this fossa in every specimen of the 2,000 temporal bones which he examined. The results of my observations showed the absence of a fossa 36 times, or 7.7%, of the specimens examined. However, in each of the 36 bones which failed to show a suprameatal fossa there were to be noticed two or more small foramina situated immediately behind the spine where the fossa should have been.

The result of my not considering these foramina as fossæ will no doubt account for the difference in the findings of Yearsley and myself.

In some cases where large foramina were present, the mucous membrane lining the underlying cells was in direct contact with the overlying periosteum and which was evidenced by the adhesion between them. In one instance a foramina of unusual size was noted. It lead directly to the mastoid antrum. It was ovoid in shape, measuring 4 mm. in its long diameter and 3 mm. in its short diameter.

The mastoid foramen is the opening on the cortex through which passes the mastoid emissary vein entering the lateral sinus at its descending or sigmoid portion. This opening may be single or double. It may be small, giving exit to a vessel of minute size, or it may be large, so that the vessel emerging from it may be a sinus carrying all the blood to the external jugular vein. The bony opening may lead directly through the cortex to the sinus groove or it may run between the outer and inner tables for a distance of as much as 15 mm. When this latter condition is present the outer opening is placed correspondingly far back. The position of the mastoid foramen varies in both a horizontal and vertical plane. Its usual location is 30 mm. behind the external meatus, and 30 mm. above the inferior end of the mastoid tip. The linea temporalis or temporal ridge or supramastoid ridge is a backward and upward prolongation of the posterior root of the zygoma. It is more or less prominent in different subjects, but it is always appreciable even in the child. Its position is almost invariably a little below the floor of the middle cerebral fossa, though in the examinations which I made to determine this I found the floor of the fossa below this line 6 times in 463 observations. This condition occurred in brachycephalic skulls.

On the inner side of the mastoid tip is the incisura mastoidea or digastric fossa, for the attachment of the digastric muscle. This fossa may be shallow or deep and occasionally its upper part is dehiscent. The absence of the superior wall was noted by me 3 times.

V. CELLULAR STRUCTURE.

The cellular structure of the mastoid process differs as to the number, size and character of the cells. According to the general character of the cells, a mastoid process may be called pneumatic,

diploic, mixed or eburnated. According to Zuckerkandel,[4] in 36.8% of the cases examined by him, the mastoid consisted, from its upper to its lower boundary, of larger or smaller pneumatic cells; in 43.2% it was formed, chiefly in its lower and posterior portion, of diploic and osseous substances; while the anterior-superior part contained pneumatic cells; in 20% the mastoid was wholly made up of diploic, less frequently of sclerotic tissue.

The results of my observations show the purely pneumatic type of mastoid process to predominate, there being 44.2%; the mixed variety, diploic below and pneumatic above, being 37.1%; and the diploic type 18.6%. It will be noted that in these figures no reference has been made to sclerosed processes, for the reason that this condition is undoubtedly always pathological, and where a process was sclerotic the observation was not recorded. Randall[5] states that he does not believe any mastoid process to consist of purely pneumatic spaces.

The cellular structure is arranged as follows: It is divided into two parts: the mastoid antrum and the surrounding cells, all of which communicate with the antrum.

The antrum is the most important of the air spaces in the mastoid process. It is constant in its presence and nearly always so in its position. It may be large or small, according to the size of the mastoid process, or it may be diminished in size by reason of osteosclerosis.

I have always found it constant, notwithstanding the claim of some observers of their inability to find it. Whiting[7] has never failed to note it in over 800 cases.

The antrum does not contain compact bony walls, they being cellular in character. The size, while uniform, may be influenced by the nature of the mastoid structure. When the surrounding bone is pneumatic the antrum is usually large, but when the surrounding bone is diploic or dense the antrum is apt to be smaller.

The average height of the antrum was found to be from 12 to 15 mm., its width about 7 mm., and its breadth 8 to 10 mm.

The roof or tegmen antri is a thin plate of bone about 1 mm. in thickness, situated externally to the prominence formed by the superior semicircular canal, and is from 3 to 6 mm. higher on a horizontal plane than the upper margin of the external bony canal. It is continuous with the tegmen covering the tympanum and aditus and separates the antrum from the middle cerebral fossa. It fre-

quently contains numerous foramina for the passage of small blood vessels. At times it has a distinctly cribiform appearance. It may be wholly or partially absorbed in old age. Burkner, as quoted by Politzer,[1] observed a dehiscence in the tegmen 167 times in a study of 765 cases. I have noted this condition 21 times.

The anterior wall may be thin, with an occasional dehiscence at its superior part so that the aditus in its vertical diameter may be as large as that of the antrum. When this does occur the facial nerve lies uncovered with bone, where the nerve turns to descend in its vertical course. This condition occurred twice in my observations.

The anterior wall separates the mastoid antrum from the bony portion of the external auditory meatus.

The posterior wall is seldom a wall, but rather a set of cells lying between it and the bony covering of the lateral sinus. These cells, which are frequently of large size, have been called by some authors the accessory antrum, but they are in reality the posterior superior cells.

The outer wall is formed by the descending plate of the squamous portion of the temporal bone. This wall may be as thin as 5 mm. or as thick as 20 mm. The average depth of the antrum from the surface of the bone is from 12 to 15 mm.

The depth differs from an age point as well as in different individuals of the same age.

The importance of the depth of the antrum lies in determining just how far one may enter with safety when operating upon the mastoid, in an attempt to reach the mastoid antrum.

Observers differ as to what is considered a safe distance to enter, when attempting to reach this cavity. Politzer[2] gives his limit of safety from 5 to 6 mm., while Schwartze[3] places his at 25 mm. The distance of the outermost part of the mastoid antrum to the external surface of the mastoid process is a much mooted point. Many observations have been made along this line with a resultant difference of opinion. (It is an exceedingly important question, since an operator feels the necessity of an approximate idea as to the depth of the antrum and to what distance he can chisel without overstepping the limit of safety.)

For the proper estimation of the depth of the antrum, some fixed and definite point should be selected from which measure-

ment should be taken. For this purpose the base of the supra-meatal spine has been generally accepted as a proper point.

I found, in my examination, that the thickness of the cortex from the outermost part of the antrum to the base of the supra-meatal spine was as follows:

Depth in mm.	Number of bones.
5	2
6	5
7	11
8	16
9	26
10	39
11	43
12	57
13	64
14	59
15	42
16	34
17	23
16	16
19	12
20	7
21	4
22	2
23	1
	463

It will be noted from these figures that the thickness of bone varies from 5 mm. to 23 mm., and that the average distance was 13.1 mm. Kerrison,[10] in a study of 30 temporal bones, measured the thickness of osseous tissue separating the antrum from the cortex and found that from the spine of Henlé that the depth varied from 6 mm. to 15 mm., with an average depth of 10.9 mm.

The character of this intervening osseous tissue may be dense, densely cellular or coarsely cellular. It was found to be dense in 251 bones, densely cellular in 8, and coarsely cellular in 124.

During the examination to determine the depth of the antrum, measurements were also made of the posterior-superior bony canal wall for the purpose of noting the relation of the depth of the an-

trum, to the length of the bony wall. The following measurements were taken:

Depth in mm.	Number of specimens.
9	2
10	7
11	18
12	31
13	50
14	56
15	69
16	73
17	54
18	36
19	27
20	16
21	10
22	8
23	4
24	2
	463

The average length of the bony canal wall was found to be 15.6 mm. In no specimen was the depth of the antrum greater than the length of the bony wall.

The cells in the mastoid process can for the purpose of convenience be designated by names according to the part of the temporal bone in which they are situated. We may have, therefore, squamous cells, zygomatic cells, tip cells, posterior tip cells, posterior superior cells, and internal petrous cells.

Many authors have classified these cells into mastoid cells, petrous cells, squamous cells and zygomatic cells. The mastoid cells include those lying below a horizontal line passing through the center of the external auditory meatus. The petrous cells include those cells which occupy the base of the petrous portion of the temporal bone and are situated above a horizontal line dividing the external auditory meatus at its center. Behind they extend to the lateral sinus, anteriorly they are limited by the premastoid lamina. The squamous cells are situated in the part of the temporal bone which contributes to the formation of the posterior wall of the external auditory meatus. The zygomatic cells are usually included under

the name of squamous cells and are to be found in contact with the meatus and bordering it, being prolonged occasionally above the meatus and in front of it into the root of the zygoma above the articulation of the jaw.

It has been my practice to designate those cells only as squamous cells which lie between the outer and inner tables of the squamous portion of the temporal bone. The tip cells, as their name indicates, are found in the tip of the mastoid process. These are usually the largest of the pneumatic cells. At times a single cell will occupy the entire tip. The posterior tip cells lie along the posterior border of the tip behind the lateral sinus. They, too, are frequently of large size. Their removal always exposes the digrastic muscle. A set of cells lying between the antrum and the knee of the sinus have been called the posterior superior cells. The internal petrous or deep petrous cells are seldom large but occupy that part of the mastoid process between the antrum and the tip cells, internal to the incisura mastoidea. The zygomatic cells are those lying above and in front of the meatus beneath the ridge at the posterior root of the zygoma, above the articulation of the jaw.

The aditus ad antrum is the connecting channel or canal which joins the antrum to the tympanum. It has an average length of from 3 to 5 mm., is from 3 to 4 mm. high, and is about 3 or 4 mm. deep.

The direction of the aditus changes from child-life to adult-life according to the position of the antrum. Its position is almost horizontal, placed directly opposite to the tympanic orifice of the Eustachian tube, but on a higher level. On the outer wall there is more or less cellular structure. Its upper wall or roof is the tegmen of the aditus continuous in front with the tympanic tegmen and in back with the tegmen of the antrum. This thin plate of bone is, as in the case of the tegmen of the antrum or tympanum, occasionally dehiscent. Its anterior end opens on the horizontal portion of the Fallopian canal. The external or horizontal semi-circular canal projects from its inner wall. Cells are frequently seen on the floor and inner wall.

The middle ear or tympanum is a small irregular air chamber situated in the petrous portion of the temporal bone. It is interposed between the external auditory meatus and internal ear. Its long axis is antero-posterior and measures from 12 to 13 mm. Its depth

2 to 4 mm., being narrower at the center of the drum. Its vertical diameter is about 13 to 14 mm., which includes the attic.

The attic, or, more properly, the recessus epitympanicus, is the highest portion of the tympanic cavity. It contains the head of the malleus and the body of the incus. It leads into the antrum by means of the aditus. The roof or tegmen tympani is a thin plate of bone continuous with the tegmen of the aditus. It separates the tympanum from the cranial cavity. In children under one year of age there is present the remains of the petro-squamosal suture. This suture may be present, in the adult, in its entirety, or remains of it recognized. When this suture exists the mucous membrane lining the tympanum is in direct contact with the dura mater. This suture was present in 12 of the ears which I examined. Cheatle[11] noted this suture 21 times in 210 temporal bones. Quite frequently a bony ridge can be noticed extending antero-posteriorly. This marks the position of the former suture.

The floor of the tympanum is also called recessus hypotympanicus. This bony plate is of variable thickness and separates the tympanic cavity from the jugular fossa. Normally there is a small foramen in the floor for the passage of a branch of the pneumogastric nerve. The floor may be partially or wholly dehiscent and when this does exist the dome of the jugular bulb presents in the tympanic cavity. This dehiscence is of very frequent occurrence in children and probably occurs in adults with much more frequency than is ordinarily supposed. I have noted this dehiscence in the adult 29 times, being complete 14 times. Koerner,[12] in an examination of 449 cases, found a dehiscence in the floor of the tympanum 30 times. Gruber[13] reports the jugular bulb cut 5 times during paracentesis due to a dehiscence in the tympanic floor.

The anterior wall is quite narrow and is deficient superiorly, inasmuch as the Eustachian tube opens into the tympanum in this situation. The orifice of the Eustachian tube is about 4 mm. above the floor. Just above the entrance is the canal for the tensor tympani muscle. Below the orifice of the Eustachian tube is a thin bony lamina situated between the tympanum and the carotid canal. This bony plate is frequently thin and even dehiscent, so that the carotid artery can be seen in the tympanic cavity. This condition I noted twice. In one instance this condition was seen by me, in the living. Lying in the floor of the bony Eustachian tube, at or near the tympanic end, are occasionally noticed some cells called

cellulæ tubaria. These cells are frequently the seat of disease and frequently are responsible for unhealed radical operations, the failure of which is usually attributed to a non-closure of the mouth of the Eustachian tube.

The posterior wall of the tympanum presents in its upper part the aditus ad antrum. Below this, on the posterior wall, is the pyramid, a cone-shaped projection which contains the stapedius muscle, the tendon of which passes through an opening at the summit of the pyramid. External to the pyramid is an opening called the iter chordæ posterius, through which the chorda tympani nerve passes. Lying on the inner wall of the typmanum are to be noted several points of importance, namely, the promontory, the oval window, the round window, the processus cochleariformis and the Fallopian canal.

VI. LATERAL SINUS.

The lateral sinus, the largest of the cranial sinuses, extends from the internal occipital protuberance to the jugular foramen, where it terminates in the internal jugular vein. The course is outward and then forward. It is divided into two parts, the horizontal and sigmoid portions. The horizontal portion situated at the base of the tentorium cerebelli, is triangular on section, with the base of the triangle being directed outward. This portion grooves the occipital and parietal bones. The sigmoid portion is situated below the tentorium cerebelli and grooves the mastoid portion of the temporal bone and the jugular process of the occipital bone. It is semi-cylindrical on section. By some anatomists it is considered as a separate sinus.

The right sinus, usually larger than the left, begins at the torcular Herophili and is a continuation of the superior longitudinal sinus. The left is the continuation of the straight sinus. The superior petrosal sinus empties posteriorly into the sigmoid portion of the lateral sinus at its origin. The mastoid emissary vein entering the cranium through the mastoid foramen also empties into the sigmoid portion of the external sinus. Numerous small veins enter the sinus from the mastoid portion of the temporal bone.

The position of the sinus in the temporal bone is uncertain. It may encroach forward on the antrum or be situated well posteriorly. It may be sharply defined or shallow.

In brachycephalic skulls the sinus is supposed to be found well forward and superficial. In dolichocephalic skulls it is supposed to be placed well back and deeply seated.

These conditions may prevail in many cases, and while many observations have been made for the purpose of determining if possible the position of the sinus by external measurements, yet I am in accord with the findings of Körner[4] and Randall,[5] who have concluded that no reliability can be placed upon external markings or measurements in determining the position of the sinus. Whiting[6] states that where the mastoid process is broad and flat, or long and pointed, the sinus is to be found far back from the meatus, and very deep beneath the cortex and diploic structure. When the mastoid process is round and narrow, or short and blunt, the sinus is situated close to the bony meatus and superficial with relation to the cortex and diploic structure.

The examination to determine the distance of the most protruding part of the lateral sinus to the suprameatal spine gave the average distance as 12 mm. The furthest away was 23 mm. Two specimens showed the sinus interposed between the antrum and the cortex. In one the sinus was in apposition to the posterior wall. The other was within 4 mm. of it.

The distance of the sinus from the spine of Henlé is as follows:

Distance in mm.	No. of cases.
0	1
4	1
6	3
7	5
8	27
9	41
10	59
11	71
12	77
13	57
14	36
15	20
16	21
17	14
18	7
19	8
20	4
21	1
23	1
	463

Amberg,[16] as a result of the examination of 17 temporal bones, found the distance of the most prominent part of the lateral sinus from the spine of Henlé to be from 6 mm. to 21 mm. He concludes that a sinus nearer than 10 mm. is displaced forward.

Ritter[17] reported a case in which the sinus touched the posterior meatal wall.

Berens[18] reported a case in which the sinus was displaced forward so that its anterior border was in contact with the posterior meatal wall, the sinus intervening between the cortex of the mastoid and the antrum.

BIBLIOGRAPHY.

1. Blake. Surgical Pathology and Treatment of Diseases of the Ear, 1906.
2. MacEwen. Pyogenic Diseases of the Brain and Spinal Cord, 1893.
3. Yearsley. *Ann. of Otol, Rhinol. and Laryngol.,* Dec., 1904.
4. Broca. Anatomie Chirurgicale et Medecine operatoire de l'oreille moyenne, 1901.
5. Randall. Amer. Text Book Eye, Ear, Nose and Throat, 1899.
6. Zuckerkandl. Handbuch der Ohrenheilk, 1892.
7. Whiting. The Modern Mastoid Operation, 1905.
8. Politzer. Diseases of the Ear, 1898.
9. Schwartze. Handbuch der Ohrenheilkunde, 1894.
10. Kerrison. *Arch, of Otology,* Vol. XXXII, No. 3.
11. Cheatle. *Jour. Laryng., Rhinol., and Otol.,* Mar., 1905.
12. Koerner. Otitische Erkrankungen, 1902.
13. Grueber. Ohrenheilk. M. F. O., 1900.
14. Koerner. *Arch. Otol.,* Vol. XVIII.
15. Randall. Transactions Amer. Otol. Soc., 1892.
16. Amberg. *Jour. Am. Med. Ass'n,* June, 1905.
17. Ritter. *Int. Central Ohrenh. No.* VI, Vol. I.
18. Berens. *Arch. Otol.,* Vol. XXVII.

PRESENTATION OF CASES.

Adeno-Carcinoma of the Maxillary Antrum.

Dr. Lee M. Hurd of New York City reported this case. The patient, a man of fifty-nine years of age, had first consulted him in December, 1905. Ten years previous to this the patient had noticed a small swelling on the alveolar process of the right side. The teeth of this side had subsequently fallen out. No symptoms other than the swelling had been noted until about a year before consultation, when the face and hard palate began to swell. When examined the growth in the mouth was found to have a cystic feel, and to encroach slightly upon the opposite side. An area of bone absorption was found below the right eye, and the growth encroached upon the infra-orbital ridge internally.

After ligating the external carotid artery the malar process was divided with a Gigli saw, the hard palate divided with the chisel, the soft palate separated, and the growth turned out. Remnants from the opposite side of the hard palate were removed, together with the bone, the cavity was packed with iodoform gauze, and the skin sutured.

The growth was pronounced malignant adeno-carcinoma by Dr. Jonathan Wright.

There were no signs of recurrence when reported, eighteen months after operation.

Primary Tuberculosis of the Septum.

Dr. Joseph H. Abraham of New York City presented a case of primary tuberculosis of the nasal septum in a woman seventy years of age. Following recovery from an attack of acute rhinitis the patient inserted her finger into the nasal cavity of the right side, and detected a swelling which she found to obstruct breathing through this nostril. Examination revealed a tumor on the anterio-inferior portion of the septum. A small portion of this was removed, and microscopical examination revealed a tuberculosis condition. There were no other signs or history of tuberculosis, nor could a positive history of syphilis be obtained.

Oedematous Nasal Polyp.

Dr. Wendell C. Phillips of New York City reported this case. The patient, a boy eight years of age, had come under his care in

October, 1905, with a history of mouth breathing for several years. There had also been occasional attacks of aural discharge, and the patient had suffered from time to time from *petit. mal*, losing consciousness for about ten seconds during each attack. Marked impairment of speech had been noted since the child began to talk. He seemed bright mentally. On October 31, 1905, the adenoids and tonsils were removed and an effort made to remove, through the anterior nares, what appeared to be a foreign body, surrounded by mucus, and situated between the left inferior turbinate and the septum far back. During these manipulations a large polypus the size of an English walnut dropped into the nasopharynx.

The tumor was submitted to Dr. Jonathan Wright, who returned the following pathological report of the microscopical examination: "This presents the unusual appearance of an oedematous nasal polyp. It contains but little stroma, a few blood vessels with considerable lowly organized tissue around them. The age, eight years, is very rare for this kind of growth. I think I have seen only one younger. (He later said none younger.) I am under the impression that when oedematous polypi do form before puberty they are more frequently attached to the inferior turbinate. After puberty they are always attached to the middle turbinate. I presume this has some relation to the physiological fact that the erectile tissue develops in the inferior turbinate mucosa and renders it insusceptible to the infiltration with serum as in the middle turbinate where are found the typical mucous polypi."

In March, 1906, patient reported considerable discharge from the nose, mostly from the left side, and inspection showed a return of the polypoid growth.

On May 8, 1907, a second pedunculated polypus, bearing histological characteristics similar to the first one, was removed from the posterior part of the left inferior turbinate. This was removed by first grasping the pedicle with forceps and tearing it loose, when the mass fell into the pharynx. This method had been found to succeed when all others seemed to fail. There had been no improvement as to frequency or severity of the attacks of *petit mal.*

Angioma.

Dr. Phillips also exhibited a little patient, eleven years old, with a gradually increasing angioma involving the entire right side of the head, from the level of the orbit to the upper portion of the neck. It had entirely separated the cartilaginous from the bony segment

of the external auditory canal, but the canal was intact and the patient's hearing good. He removed some redundant tissue from the concha, which had shown the growth to be angiomatous in character. There was great deformity on account of the drooping of the ear upon the affected side. A fuller report had been made of this case to the Section on Otology of the New York Academy of Medicine, which would appear, with the discussion, in the transactions of that body.

Two Cases of Otitis Media Catarrhalis Chronica Showing Improved Hearing After Acute Mastoiditis Treated by Operation.

Dr. William Sohier Bryant of New York City reported these cases. In the first patient, aged nineteen years, the hearing for the watch, previous to mastoiditis, was fifty inches in each ear. Since the convalescence from mastoiditis two years ago, the hearing had continually improved in the operated ear, and continually decreased in the non-operated ear. The hearing when reported was thirty-six inches in the non-operated ear and 7½ feet in the operated ear.

The second case was that of a child, thirteen years of age, who had had chronic middle ear catarrh and mastoiditis, having been operated upon for the latter. Before the mastoiditis the hearing was between twenty and twenty-six inches for the watch in the non-operated ear; in the operated ear slightly less. Two months after operation, the hearing remained the same in the non-operated ear and rose to twelve feet for the same watch in the operated ear.

Cleft Palate.

Dr. Truman M. Brophy of Chicago presented a case illustrative of the value of early operation for the closure of cleft palate. When the patient was three months old the hard palate was brought together, the lip was closed when the child was five months old, and the final operation for closure of the soft palate was performed at the age of two and one-half years. Believing cleft palate to be due to a wedging process which takes place in embryo, and that the bones are not lost but merely separated, his operation was designed to supplement the work of Nature by bringing these bones together and moulding them in place.

Laryngeal Tuberculosis.

Dr. Robert Levy of Denver, Colo., presented a patient who, for two years, had been suffering from pulmonary tuberculosis, and

for one year and a half from laryngeal tuberculosis, the active manifestations being on the internal surface of the left arytenoid cartilage, with characteristic oedematous infiltration of both arytenoids, and aphonia. After three months' treatment the voice improved. The ulceration healed entirely. When reported there was still some infiltration, but the condition was improving from day to day, despite many recurrent infections.

A Case of Angio-Neurotic Edema of the Throat.

Dr. Thomas H. Halsted of Syracuse, N. Y., presented this case. When he first saw the patient, a young woman, there was a leathery thickening of the soft palate, the throat was edematous, the arytenoid cartilages were thickened, and the vocal cords were enormous. She complained of slight dyspnea. In the middle meatus on the right side there was slight necrosis and a little pus. He thought the condition due either to an inherited syphilitic process or to tuberculosis. After repeated negative examinations of the sputum tuberculosis was excluded. The patient was then put on anti-syphilitic treatment, which had been continued for the past two years. There had been no essential change in the throat, the edematous infiltration remaining the same. The dyspnea had decreased. There was no apparant loss of bone in the nose, but on the septum there was some superficial loss of tissue. At six years of age the patient had had scarlet fever, with scrofulous glands in the neck, from which she recovered. Some years later she had had necrosis of the ankle, which healed after curettage. So far as she knew there was no history of syphilis on either side, nor was there any history of tuberculosis. Two months ago she had had what appeared to be erysipelas of the face and scalp. It had occurred to him that the condition of the throat was angio-neurotic edema, of which Osler, Bloodgood and others had reported cases.

PRESENTATION OF INSTRUMENTS, ETC.

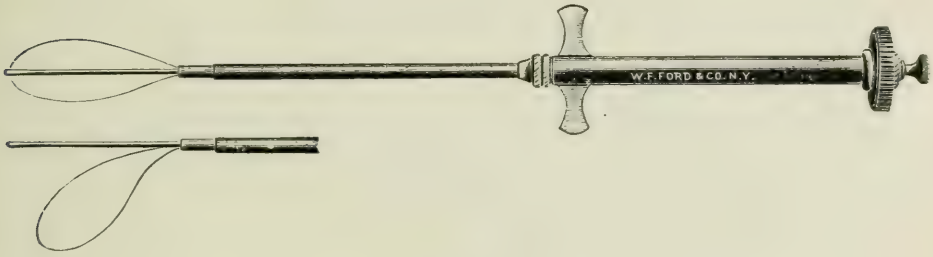
Safety-Pin Closer.

Dr. Chevalier Jackson of Pittsburg, Pa., presented a modification of Dr. Mosher's Safety-pin Closer. The latter consists of a stem with a ring attached, in conjunction with which is used a second instrument in the form of a fork or prong. With the fork the pin is pushed into the ring and closed. In unskilled hands difficulty is experienced in getting the ring down past the safety-pin in the oesophagus or trachea. The modification obviates this diffi-

culty by having the ring in the same place as the stem when introduced; having passed the safety-pin, the ring is moved to a right angle by means of the lever handle.

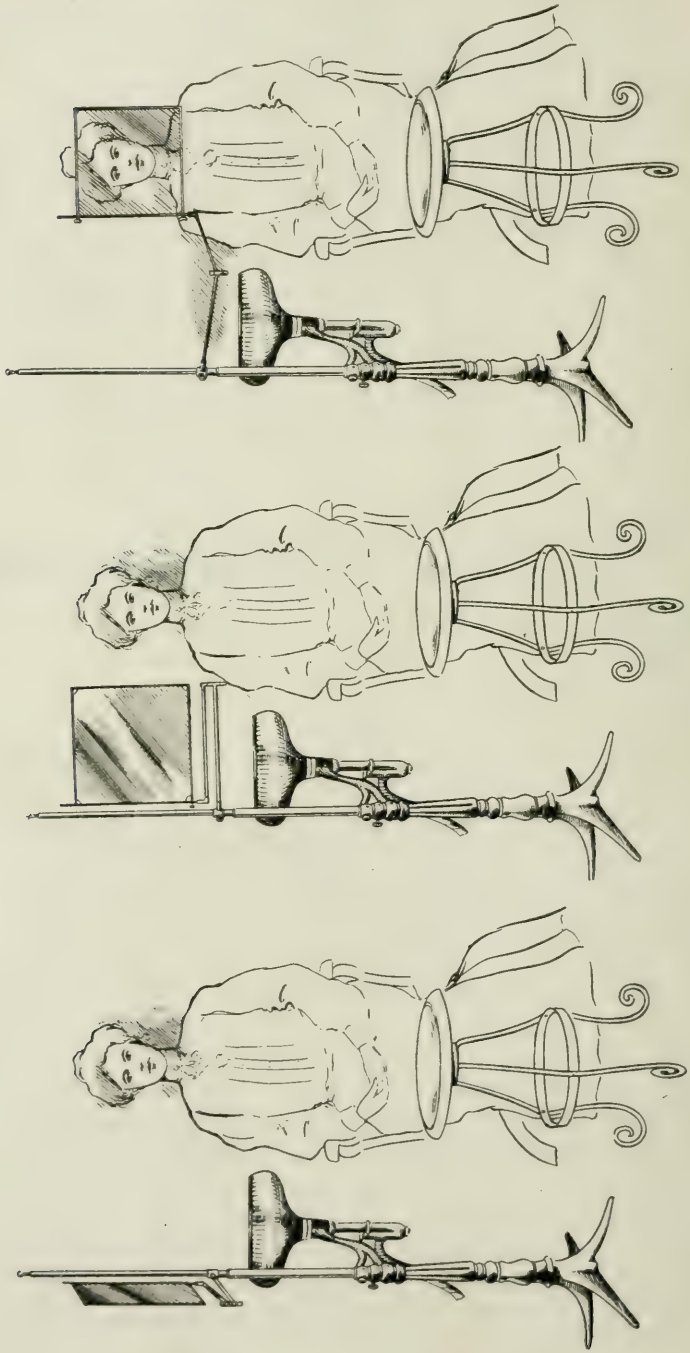
Anti-Tubercle Screen.

Dr. Robert Levy, of Denver, Colo., presented a glass screen to be placed between the patient and the operator, especially in the treatment of tuberculosis cases. The accompanying cut demonstrates the simplicity of the apparatus and the ease with which it may be readily placed before the patient and quickly moved aside. It may be attached to the standard of the cuspidor, to a separate standard or to the adjoining wall. The advantages claimed for this screen over others, which have from time to time been devised, are its simplicity, its cheapness, and the ease with which it may be manipulated.



A Snare.

Dr. L. L. Mial of New York City presented a snare (see cut) which he had designed for nasal use, especially in those instances where it is necessary to have the wire loop turn at an angle after insertion in the nasal chamber. To do this the natural elasticity of the wire is made use of and the loop can be made to turn from a very obtuse angle to even more than a right angle. The instrument consists of the ordinary canula, a hollow draw-bar to which the loop is attached with regular screw on opposite end, and through this hollow draw-bar a long stylet passes and projects at the distal end sufficiently to hold the loop straight after it has been bent to the desired angle. In removing a posterior tip the wire is first fastened to the draw-bar and then drawn slightly into the canula to secure it; the loop is then forcibly bent to the angle desired, and the stylet is passed through and the loop is held in straight position by hitching it over the tip. The loop is then passed along the floor of the nose until the posterior wall of the pharynx is reached.



The stylet is then withdrawn and the loop being released turns at the angle to which it has been previously bent, and engages the hypertrophy very promptly. He had used it for several months with great satisfaction. The instrument is made by the Ford Surgical Instrument Co., of New York City.

PRESENTATION OF PATHOLOGICAL EXHIBIT.

Anomalies of the Accessory Sinuses.

The anatomical exhibit consisted of three sectional skulls, each specimen beautifully demonstrating one or more anomalies of the accessory sinuses.

The first specimen (loaned by Dr. Abraham) demonstrates one large ethmoidal cell occupying almost the entire ethmoidal labyrinth. It measures 2.3 cm. antero-posteriorly, 1.1 cm. laterally, and 1.5 cm. in its vertical diameter. In front of this cell is a smaller cell, which projects into the frontal sinus posteriorly, constituting a so-called frontal bulla. This cell is 1 cm. long, 0.6 cm. wide, and 0.5 cm. high. Internal and anterior to the hiatus semilunaris, is a so-called infundibular cell, representing the anterior expansion of the unciform process. This cell causes a projection in the floor of the frontal sinus. The ostium of the frontal sinus is not at the lowest point, but about 0.6 cm. above the lowest point of its floor.

The second specimen (loaned by Dr. Abraham) demonstrated an exaggerated deflection of the septum between the frontal sinuses. Even at the lowest part of the sinus, where the position of the septum is considered to be always constant, the septum is displaced 5 mm. to the left. From here it goes upward and outward, so that in its upper part, the septum is 1.5 cm. to the left of the median line. The right frontal sinus is about five times the size of the left sinus. Both sinuses show prolongations laterally, to a point 1.5 cm. external to the supraorbital notch, and backward, into the orbital plate. The right sinus has only one small vertical septal ridge in its posterior angle. The left sinus is subdivided by three fairly large incomplete septa.

The third specimen, shown by Dr. Abraham, was loaned by Dr. J. W. Gleitsman. It shows a very large frontal sinus, which extends upward about 2 cm. into the vertical portion of the frontal bone, outward to the articulation with the zygoma, and backward into the orbital process of the frontal bone about 1½ cm. It covers in the anterior ethmoidal labyrinth, above. There is a marked prominence

on its posterior and internal walls, caused by the presence of a so-called frontal bulla. This frontal bulla is nothing more nor less than a large posterior ethmoidal cell, which lies just behind and internal to the frontal sinus, and opens into the superior meatus. The anterior ethmoidal labyrinth comprises one large cell, which is represented by the ethmoidal bulla. The bulla forms a very marked prominence on the outer wall of the middle meatus. The posterior ethmoidal labyrinth consists of one large and four smaller cells, each opening by a separate ostium into the superior meatus, in addition to the frontal bulla above described. The sphenoidal sinus is very large. It measures 3½ cm. in its antero-posterior diameter. It extends for a considerable distance forward into the lesser wing, outward, into the greater wing, and backward, into the basilar process of the occipital. It has a large ostium, about 6 mm. in diameter, opening into the spheno-ethmoidal recess. The outer wall of the inferior meatus shows a marked outward bulge, so that the inner and outer walls of the antrum are almost in contact. An opening drilled through the alveolus, would enter the inferior meatus, instead of the antrum.

Tubercular Tumor of the Nasal Septum.

Dr. Abraham exhibited a section made by Dr. Jeffries from a tumor of the nasal septum in a woman of seventy. Dr. Jeffries made the following report: "The tissue from the nose marked J. C. received from you, is typical tubercular tissue with multiple tubercles, most of which are undergoing cheesy degeneration and contain giant cells. The nodules are of epitheliod cells and are surrounded by zones of exudative infiltration."

Laryngeal Tumor.

Tumor of the Middle Turbinate.

Fibro-myxoma of the Upper Jaw.

Dr. E. G. Zabriskie exhibited three pathological specimens, from the laboratory of the Manhattan Eye, Ear and Throat Hospital.

The first was a section from a tumor sent by Dr. Chevalier Jackson, which filled the whole right side of the larynx, and involved the left ventricular band. It resembled very closely the cancroids frequently seen about the face. Scattered throughout a fibrous stroma, there were patches of aberrant epithelium, whose centres had undergone hyaline degeneration, and in places had dropped out, leaving a rim of atypical cells, which looked like a glandular

acinus. There were several more or less typical pearls or whorls scattered throughout the section. The cells had a rather granular protoplasm, and showed a decided lack of irregular karyokinesis.

The second section was from a growth of the middle turbinate, sent by Dr. Smith. The patient's age is sixty-five; duration, four years; recurrence for the third time. The glandular elements of the mucosa are considerably increased, although not enough to warrant a diagnosis of pure adenoma, but the most striking changes occur in the connective tissue trabeculae, which are remarkably hyperplastic, and show a very curious hyaloid degeneration. This often appears among the glandular elements in the form of homogeneous, glistening droplets or facetted particles, numbering four to eight, and held together by a cell membrane. This has been fully described by Dr. Wright in the American Journal of Medical Sciences.

The third was a section of a fibro-myxoma of ordinary type, springing from the alveolar process of the upper jaw, involving the antrum of Highmore, and the maxillary ridge, reported by Dr. Haskin.

Sarcoma of the Antrum.

Dr. Price-Brown exhibited a pathologic specimen, weighing nearly two ounces, which he had removed from the left maxillary antrum, through the anterior wall. Duration of illness six months. During this period, many polypi were removed from the nose and the antrum opened through the inferior meatus and the last alveolus. The maxillary protrusion constanly became larger. A radical operation was done by Dr. Brown. The outer wall of the antrum was largely destroyed. A large growth filled the antrum, and was shelled out without much difficulty. The cavity was curetted and packed with iodoform gauze. Microscopical examination showed the growth to be a myxo-fibroma, with a few suspicious cells, possibly sarcomatous. The wound in the maxilla closed, but the appearance was not satisfactory.

Further report: A second section of the tumor was found to contain spindle-cells, making the diagnosis that of sarcoma. As the protrusion of adventitious tissue from the jaw rapidly increased, pressing out the cheek very noticeably, the reporter, on the 26th of June, removed the greater part of the affected maxilla. There was much hemorrhage, but the patient rallied well.

MEETING OF EASTERN SECTION, HELD IN PROVIDENCE, RHODE ISLAND, JANUARY 5, 1907.

PAPER:

A CASE OF SARCOMA OF THE MAXILLARY SINUS.

F. T. ROGERS, M. D.

I desire to present this case to the section for two reasons; first, to recite briefly the remedial measures which have been followed in a peculiarly distressing case and to show the condition of the growth after seven months of active treatment and to afford an opportunity to examine a case which is not unusually rare but presents some peculiar features, and second, to gain from the discussion that may follow the ideas of the members as to the advisability of further procedure.

This patient has been under my observation for a matter of fifteen years. Specific disease in early life destroyed the septum but for a number of years there has been no recurrence of the disease. In May last he came to me with a swelling in the left maxillary region. Anticipating an empyema of the sinus, a perforation was made through the nose but nothing was obtained and later an opening was made into the antrum through the canine fossa and the present condition was found. At that time the cavity was thoroughly curetted. The growth apparently sprang from the anterior lateral wall of the sinus and was crowded over so as to completely destroy the integrity of the nose. The sinuses of the nose were exposed both to the finger and to the mirror. The inferior orbital wall had been perforated by the pressure of the growth and a considerable quantity protruded into the orbital cavity. This was also removed with the curette as completely as possible. There was no recurrence until some time in July when it rapidly began to grow worse, and early in the summer, by my advice, he consulted Dr. Coakley who gave as his

opinion, that the complete abolition of the left side of the face was the only opportunity which presented itself for relief of the trouble but that he did not advise it. Later it was decided to attempt the removal of the superior maxillary bone and the operation was started with that intent. The extensive destruction of tissue, however, which was encountered, and the probable loss of the integrity of the eye and the hideous deformity which would have resulted, caused us to hesitate and after the bone had been exposed by the ordinary incision it was decided to clean the cavity as well as possible and allow the case to progress without further surgical interference. The wound healed kindly with the exception of the cicatricial attachments which formed under the edge of the orbit but about the second or third week it took on an exceedingly offensive odor and the growth has gone on with intervals of apparent quiescence. There is no pain and there has been very little disturbance of the general health and the condition which shows today is such that I would like you all to see. The treatment which has been followed comprises all the methods which are known to me by personal experience and from my study of the literature on the subject. He has been taking methylene blue combined with quinine and belladonna for several months. This idea was taken from Dr. Jacobi's monograph on the value of the fluorescent salts in sarcoma. The X-ray was tried with no other effect than to destroy the unpleasant odor. The Finsen ray was experimentally used with the effect of producing an erythema of the skin and undoubtedly aiding in the formation of a local abscess which followed the injection of trypsin. He has had trypsin injections up to the limit of the ampoule which contains 20 minims beginning with the smaller doses, injected into the arms, shoulder and buttocks, and small quantities were injected deeply into the growth and into the skin about the growth. One unfortunate injection in the face caused a needle abscess which gave rise to considerable pain for some days but which is the only pain he has suffered. Lately I have used the clear injectio-trypsini without diluting it, injecting it into the opening which is left in the cheek. I think, without exception, that this procedure has shown the most marked effect of any remedy which I have used. For the last month or six weeks the growth has not increased and the odor has disappeared, and it seems to me that it is in a measure smaller than it was

early in December. Several times, when the growth has encroached upon the mouth it has been necessary to cut it away in order to prevent interference with swallowing and with the mastication of food, and each time that the growth has been mutilated in any way there has been an increase in the formation, very marked within the week or ten days following.

As I said before, it is not an unusual case, yet it is not so common that we can afford to neglect it and I shall be very glad to show the patient to those who are interested and later to learn your opinion as to what should further be done in the case.

Dr. Dench—May I ask Dr. Rogers if he has used radium tubes at all?

Dr. Rogers—I have not. I did not use Coley's fluid because of a very unfortunate experience in a case of sarcoma and the difficulty which I had in getting the reaction which I expected. Nothing has been said about the use of that remedy which has been reported as influencing the growth. I gave this man two injections of adrenalin chloride. The first was injected into the tumor and caused the most intense pallor that I have ever seen in any living man and a disturbance of his mental faculties. In about ten minutes he gradually recovered. The second one I injected very slowly and before I had finished emptying the syringe he fell from the chair and I thought he was dead. That condition continued for nearly an hour before I was able to remove him from the floor where he had fallen. In about ten minutes perspiration broke out which had a most offensive odor. Those are things which I would not try again.

PAPER:

PROF. GUSTAV KILLIAN AND HIS WORK. EXPERIENCES OF A RECENT VISIT TO FREIBURG, GERMANY.

WILLIAM E. CHENERY, M. D.

Freiburg, sometimes called the "Pearl of Breisgau," is a beautiful city, situated on the borders of the famous Black Forest in southwest Germany and south of Heidelberg. It is a manufacturing town of about 76,000 and is an old and important educational center, famous for its university, which has about 2,000 students, more even than Heidelberg.

The busy laryngologist on a short vacation tour of inspection is not so much interested in the beautiful walks and drives around Freiburg, the Schlossberg or the miraculous spring of St. Ottilien, the noted Gothic Cathedral or the curious old town gates. To him the central attraction is one who for years has been foremost in the advancement of our specialty, Dr. Gustav Killian, Professor of Laryngology and Rhinology, in the University of Freiburg. Prof. Killian's achievements are well known, but perhaps it would not be out of place to recall some of his special work. You are familiar with his method of examining the larynx, which makes possible a view of the posterior laryngeal wall, and subglottic area. By this method a good view of the trachea to the bifurcation is often obtained. Guisez says: "C'est Killian qui est le veritable createur de la tracheo-bronchoscopie."

Kussmaul attempted direct oesophagoscopy in 1868. While Mackenzie and Mikulicz, Hacker and Rosenheim had developed the method, and had removed foreign bodies by means of tubes before 1895, Killian in that year showed the possibilities of the use of the bronchoscope. In March, 1897, he removed a bone from the trachea. This led to the exploration of the bronchi, and on July 24, 1897, he showed to the medical society that he was able to introduce tubes freely into the air passages. By his experiments he learned that the bronchi were extensible and freely movable. This

made possible the adoption of superior bronchoscopy. He also found by turning the head of the patient, he was able to explore the right or left ramifications of the bronchi.

In September, 1899, he first reported his method of submucous window resection of the nasal septum, which operation has been perfected by Killian and our own rhinologists, until now the submucous resection is by many considered the best, and by some, the only operation for septal deviations.

In 1902, he published a description of his radical operation on the frontal sinus. Besides, Killian has done much research work in embryology and in the comparative anatomy of the nose and nasal cavities. The results were published in 1904 in his "The accessory sinuses of the nose and their relation to the neighboring parts." He has devised many instruments. His speculum and submucous instruments are well known; and his bronchoscopic instruments are numerous. The right-angled trocar for washing the antrum of Highmore, the "V"-shaped chisels and eye protector for the frontal sinus operation, his sphenoidal punch, not to mention many other instruments, show his mechanical genius. He has written many articles for the German, French and English journals; besides he has made numerous charts and models. I remember the pride with which he showed me a very large papier-maché model of the maxillary bone, with the turbinates attached, which he used for instruction purposes.

In the latter part of August it was my privilege to be in Freiburg and be a visitor in Killian's clinic. He was cordial in his greeting and seemed to be pleased to have Americans visit him. His keen eye and quiet manner immediately betoken the real student. He is about fifty years of age. He speaks English fairly well. However, after a few pleasant words, or answering questions, he is apt to retire to his private office, which adjoins the general clinic room, leaving the visitor in the care of his first assistant, Dr. Karl Von Eicken, who is very genial and affable. Von Eicken speaks English fluently and takes great interest in explaining Killian's methods. As an assistant, he is under a five years contract at a nominal salary, and during that time can not practice privately. This custom is most excellent, for it assures the master of a valuable assistant, and makes possible much research work.

What can one accomplish in a short time in Killian's clinic? By arranging before hand as to time, one can take a very instructive and practical course in bronchoscopy and œsophagoscopy. Many

of us have had the opportunity of taking Dr. Mosher's most excellent course, and practiced passing the bronchoscope and œsophagoscope on the cadaver. In Freiburg, one learns the technique, and actually passes the tubes on the living. To see the ease with which it may be done, without a general anæsthetic, is very interesting and shows the possibilities of a larger use in the future.

The course includes the history of tracheo-bronchoscopy and œsophagoscopy, the use of Kirstein's head lamp, which Killian uses; and practice in diagnosing foreign bodies at the end of different length tubes. Killian does not use the tubes with a light near the end. Having become proficient in this, inferior bronchoscopy is practiced, with 20% cocaine as the local anaesthetic. After entering, at right angles, the tracheotomy wound, the tube is passed downward to the bifurcation, when cocaine must be again applied. The patient sits on a low stool and the operator stands directly in front of him. After thoroughly cocainizing the bifurcation, the exploration may proceed to the bronchi, care being taken to be gentle in the manipulation.

Superior bronchoscopy is then practiced, the parts being well cocainized with 20% cocaine. The divided Kirstein autoscope is used to assist in the introduction of the tube, until past the vocal cords, when it is removed and the examination proceeds as in inferior bronchoscopy, the position of the patient and surgeon being the same. If a general anæsthetic be given, Rowe's position is used, and the surgeon stands at the patient's head and facing his feet.

The last exercise is in œsophagoscopy, which is performed with the aid of 20% cocaine, applied as before, to the pharynx and fauces, and base of tongue and arytenoids, with a swab. To assist in introducing the instrument, the finger is used as a guide, and a flexible bougie is introduced through the tube about two inches. The instrument must be well oiled. The cricoid is the greatest obstacle in the passage of the instrument. Force must not be used. When past this point the bougie may be withdrawn and the instrument passed downward, at the same time inspecting the walls of the œsophagus.

The endoscope and cystoscope have been used with much success in other parts of the body. Because a few accidents occurred in earlier years from crude instruments and faulty technique, direct examination of the œsophagus and trachea have been avoided. But Mikulicz long ago said every normal man could be examined with the œsophagoscope. Prof. Stark says, passing the œsophagoscope

depends more on the skill of the examiner, not on the anatomical build of the patient. Great improvement has been made; and since 1900, hundreds of examinations have been made without accident.

The laryngologist will be called on more frequently not only to remove foreign bodies from the trachea, bronchi and œsophagus but also to treat medically and surgically, these parts. In the same line, Dr. Chevalier Jackson has shown us that direct inspection and treatment by means of the gastroscope, is within the province of the skilled laryngologist. What we need is practice. More attention to this subject should be given in our medical schools. The use of these instruments is applicable to ulcerations of tuberculosis, syphilis, and beginning cancer; to direct examination of tumors, benign and malignant; to diverticuli and stenoses from various causes; to neuroses, as well as foreign bodies. Also by giving the exact location of disease, proper surgical procedure may be aided.

Killian has removed a sarcoma in a man of sixty by the natural passages. It is contraindicated in extremely nervous people, in advanced age, where there is a rigid vertebra, in extreme cachexia, and advanced tuberculosis; also in emphysema and acute inflammatory processes of the lungs; in aneurism of the aorta, and in organic heart disease. The difficulties of superior bronchoscopy and œsophagoscopy are in having a good set of teeth and a short, thick-set neck.

To one who is used to the large clinics of our hospitals, the clinic at Freiburg seems small; during my stay, there were not more than ten to fifteen patients per day. From one who spent several months in Freiburg, I learned there were never more than twenty to twenty-five. From the fame of its clinician, however, material is drawn from a very wide circle.

A few points noted in the frontal sinus operation I saw Killian do may be of interest. The patient was a man of about 45 years, with left chronic frontal sinusitis, previously operated on by the Ogston-Luc method, with only temporary relief from pain and suppuration. The patient had come many miles for operation by Prof. Killian. A careful intra-nasal examination and X-ray showed the presence of the disease and extent of the sinus. Ether was the anæsthetic. No post-nasal plug was used, only the three cigar-shaped pieces of cotton packed through the anterior nares. The head of the patient was slightly raised during the operation. The eye-brow was not shaven. Great care was taken to preserve intact the periosteum over the narrow bridge, and the orbit was protected

well by an assistant, with Killian's orbit guard and retractor. The opening below the bridge was made as large as possible, and great care was taken in exploring the whole of the sinus, especially the orbital portion, and all septa were removed. All the ethmoidal cells were carefully destroyed. The wound was closed by aluminum bronze sutures. The nose was lightly packed with gauze for twenty-four or forty-eight hours. Light compression was made over the frontal to make the tissues sink into the sinus cavity. The patient, afterward, was not allowed to blow the nose, but was instructed to sniff back into the throat all secretion. Crusts were removed from the anterior nares by forceps. Several cases radically operated on some time before were seen, and the results were very satisfactory, relief from pain, cessation of suppuration, and only slight deformity. One man, who had a very large sinus, and so more deformity, received a second injection of cold paraffin while I was there, completely filling out the depression.

All over Germany severe operations are being done with injections of cocaine and adrenalin, which we would consider must be done under general narcosis. I saw several mastoids, an excision of the jaw, and several radical Caldwell-Luc operations on the antrum, all done under cocaine anæsthesia. One day, in Killian's clinic, a young man, 21 years of age, had a Caldwell-Luc operation on his antrum, sitting upright, with only the use of cocaine and adrenalin. At the end of the operation the young man was a little faint. After lying down for a few minutes, he walked back to his room in another hospital building; no packing having been put into the antrum. He did very well, and had no post operative hemorrhage.

Killian is a thorough anatomist, an experienced and resourceful surgeon. Careful and thorough in his operations. A man justly famous. A worthy guest of our next annual meeting.

PAPER:

THYMIC TRACHEO-STENOSIS. TRACHEOSCOPY. THYMECTOMY. CURE.

CHEVALIER JACKSON, M.D.

Much of the literature on the diseases of the thymus gland is devoted to theoretical arguments as to the cause of "thymus death" (thymustod). A child dies suddenly with more or less stridor and cyanosis, and nothing but an enlarged thymus gland is found at autopsy. The gland, however, does not seem to press upon the trachea. The authors then speculate upon theories as to pressure on the pneumogastric, pressure on the pulmonary artery, "hyperthymisation of the blood," irritation of the large vessels and nerves by the friction of the to and fro movements of respiration, paralysis of the vaso-constrictors, status lymphaticus, etc. Theoretic arguments and anatomic demonstrations are brought forward to show the softness of the gland, and the relative incompressibility of the trachea.

It has been my privilege, with the aid of the bronchoscope, to demonstrate beyond all doubt upon the living patient the purely mechanical nature of "thymic asthma" in one instance. This of course does not prove that every case has this same pathologic mechanism, but it does prove the occurrence of that which many, including Friedleben, von Kundrat, d'Escherich and Paltauf have denied, namely, that a hypertrophic thymus can compress the trachea sufficiently to obliterate its lumen. It does disprove Friedleben's dictum—*es giebt kein asthma thymicum*, though it would seem more accurate to call it thymic tracheo-stenosis.

In view of my tracheoscopic findings, it is easy to understand sudden deaths from thymic hyperplasia, for we all know how suddenly the end comes in any form of tracheal stenosis, and how impossible it is to get respiration started after it has stopped when there is even slight obstruction, unless an instantaneous tracheotomy be done. In these thymic cases a long canula reach-

ing below the obstruction would have to be inserted. With a tracheal lumen diminished to a chink, the slightest turgescence, as from a cough, would be sufficient to cause temporary swelling, and momentarily to shut off the passage of air. The coughing and gagging during an examination would do the same thing. Once the air is shut off the usual phenomena of asphyxia are sufficient to engorge the thymus gland and all the vessels passing through the upper thoracic opening, which prolongs the obliteration of the tracheal lumen until death supervenes. Then the recession of the blood and the sagging of the viscera, together with the usual autopsic technic, would serve to allow the trachea to assume its normal lumen. Thus the pathologist finds the gland hypertrophied but not compressing the trachea.

Earl L., age 4. Admitted to the Western Pennsylvania Hospital in the service of Dr. Ogden M. Edwards, August 30th, with the following history:

Child is the youngest of eight; one brother died a year ago with typhoid fever, another with tuberculosis. Two others died in infancy. This boy has always been well, except for a mild febrile attack a few months ago; not bedfast.

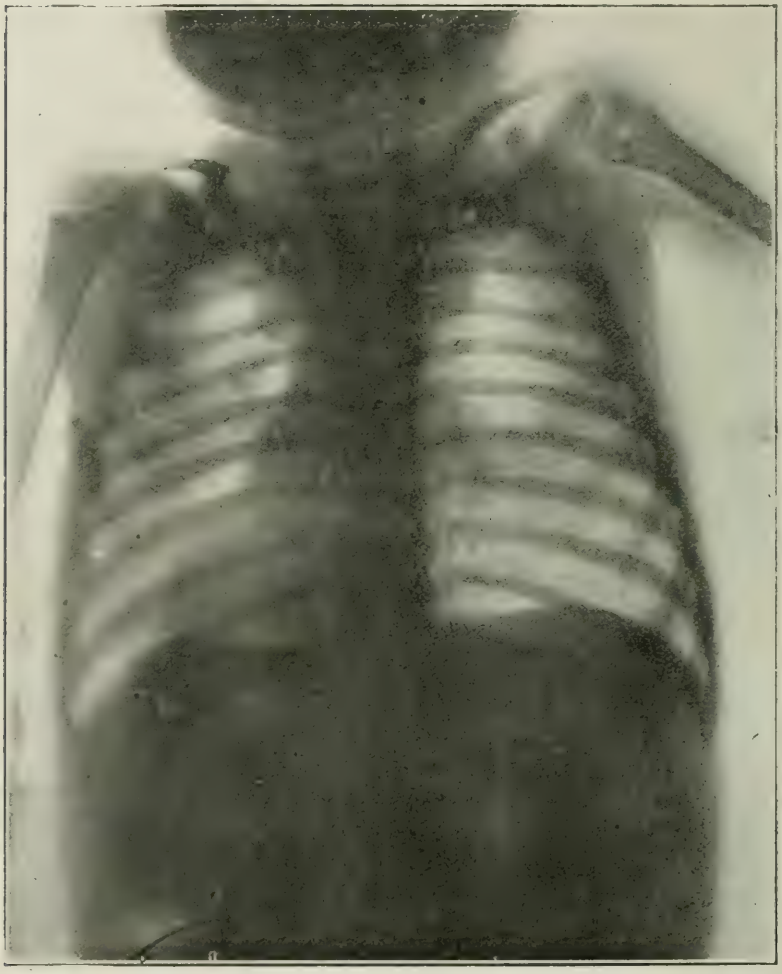
Six weeks before admission went to bed apparently well; wakened in the night by a sudden croupy attack. Since that time difficulty in breathing has been growing steadily worse and is now very marked and stridorous.

Report of examination by Dr. John W. Boyce: "Child manifests considerable expiratory dyspnoea of obstructive type, made worse by sitting erect. Good color, and a fair general appearance. No signs of 'lymphatic constitution'. Pulse rapid, but of good quality. Tympanitic note over sternum and at sides extending farther to right than left. Over same area increased voice conduction, tracheal breathing, and a whistling tracheal rale. Over rest of lungs normal puerile breath—sounds the same on both sides. Diagnosis:—Obstruction in trachea. Tubes dry, lungs normal."

I was called to the case and finding the larynx free from obstruction, suspected a foreign body, and ordered a radiograph. A radiograph by Dr. R. H. Boggs showed the shadow of an enlarged thymus. (Fig. 1.)

Operation September 5th. Assisted by Dr. J. J. Schoenig I opened the trachea under infiltration anesthesia, the rings seem-

ing of normal resiliency. The thymus gland was seen to extend abnormally high in the neck, reaching to the thyroid gland, and rose somewhat when the patient coughed. On passing one of my bronchoscopes I discovered a scabbard trachea with a chink of not



over 2 m.m. on inspiration and 1 m.m. on expiration, the obstruction extending from the second to the fourth ribs. The tracheal mucosa was collapsed from before backward almost into contact. The collapsed walls opened up ahead of the tracheoscope not unlike the cervical esophagus, and like the esophagus, it tended to open on

inspiration. The mucosa looked slightly congested, but was not edematous.

The passage of the bronchoscope relieved the dyspnoea instantly. When the bronchoscope was withdrawn no air entered and asphyxia threatened. A long tracheal canula was inserted in the wound, giving partial relief. From measurements taken with the tracheoscope, I had made a special long tracheotomy canula extending to within one c.m. of the bifurcation. This gave absolute relief and was worn four weeks, until the operation on the gland could be done.

The day after the tracheotomy, discharge from the tracheal wound was very profuse. Numerous moist rales of large size were heard all over both lungs. Within a few days these diminished, as expectoration of purulent material became more profuse. There was no bronchitis, (Boyce) the rales being produced by pus that had run down from the tracheotomy wound.

On Sept. 8th the child developed a mild tonsillar diphtheria, for which he was transferred to an isolation cottage and placed under the skilful care of Dr. G. W. Grier. On the 9th, 3000 units of antitoxin were given, and on the 10th, 1500 more. On the 11th, membrane had almost entirely disappeared.

After a negative culture the child was released from quarantine on Sept. 28th, and was found to have a mild bronchitis of the middle sized tubes. There was some pallor and a rapid pulse. The abdomen was quite tympanitic and the stools were of foul odor. All these intercurrent affections deferred operation until Oct. 2nd. During these four weeks the breathing was normal, except, of course, the usual stridor serraticus. Sept. 28th, blood examination showed haemoglobin 75 per cent; red cells 6,780,000; white, 18,000; polynuclear, 73 per cent; small mononuclear 18 per cent; large mononuclear, 3 per cent; transitional, 8-10 per cent; eosinophile, 3 8-10 per cent.

Operation Oct. 2nd, assistants, Drs. J. H. McCready, J. B. Penrose, and John W. Boyce. Chloroform was skilfully administered through the tracheal canula by Dr. I. H. Alexander. Upon removing the canula for tracheoscopic examination, absolute stenosis followed, owing to collapse of the trachea. The tracheoscope showed a mild acute tracheitis, without edema or ulceration. The canula was then re-inserted. A curved transverse incision was made just below the upper border of the sternum to

avoid the tracheal wound. The skin was retracted upward, and the incision carried throughout down to the thymus gland, the sternal attachments of both sterno-mastoids being severed. The thymus bulged into the wound with each coughing effort. I passed my finger down into the mediastinum, breaking up numerous fine fibrous attachments to sternum, care being taken to not wound the pleura or pericardium. The gland was gradually drawn up with tissue forceps until its tip was turned out of the wound, when the tip was seen to be drawn in with each inspiration. Several fibrous attachments were ligated and divided. The lower extremity of the gland was ligated and removed. These manipulations with the finger in the mediastinum were rendered possible by the tracheal canula, which prevented compression of the trachea. While the finger was in the mediastinum it was noted that the lower trachea rings were of normal resiliency. All ligatures were attached to the upper border of the sternum and one transverse stitch was passed through the pre-tracheal fascia, and secured to the manubrial periosteum. The sterno-mastoid tendons were sutured into position. Silk worm drainage was passed from end to end of the wound and the skin was sutured. A wet bichloride dressing was applied covered with rubber tissue, and then a bib of oiled silk was placed under the tape holder of the tracheal canula to try to prevent the secretions coughed up from infecting the wound. Child had stood operation fairly well, but showed some weakening and rapidity of pulse, while the finger was in the anterior mediastinum.

"Patient passed a fair night. Pulse very rapid (over 150) but of good volume. Breathing rapid and irregular. Rattling of tracheal pus prevented satisfactory examination of chest." (Boyce).

The attempt to prevent infection of the wound by the tracheal discharge proved futile, as the two wounds were less than 1 c.m. apart. The thymic wound healed kindly by granulation, leaving a depressed scar. At each dressing bubbles of clear serum were seen exuding from the mediastinum on expiration, and a probe could be passed downward readily for a distance of three centimeters, but strange as it may seem, this cavity never became infected, which was due, I think, to dressing the wound with wet bichloride gauze every three hours. This kept the entire wound free from pus. The tracheal canula was removed on the eighth

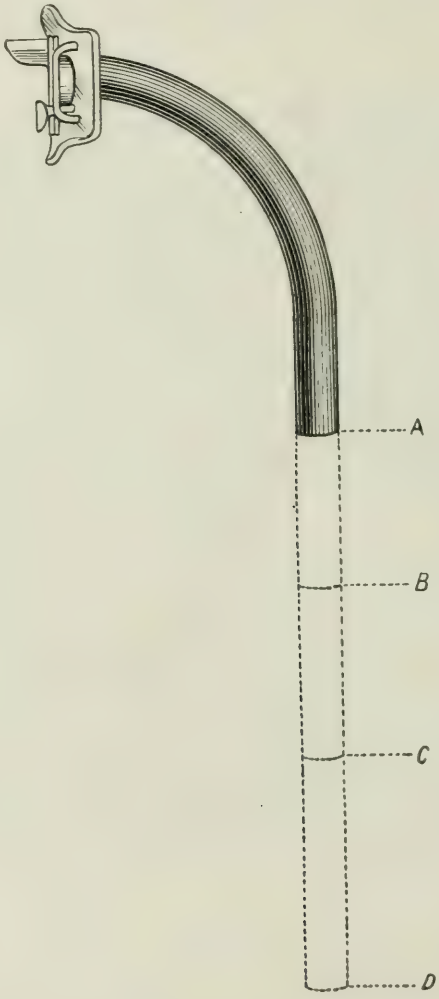
day after operation. The boy made a slow recovery, due to the intercurrent affections, in my opinion, rather than to the operation. He never showed the slightest stridulous sound after the thymectomy. His last blood examination, three months later, showed red cells 6,440,000; white cells 12,480; hemoglobin 85 per cent. He was plump and well nourished. (Fig. 2.)

Dr. Joseph H. Barach examined the gland histologically and reported as follows:

"Section of gland specimen showed it to be made up of about equal parts of thymus gland tissues and changed-fibrous tissue. Half of the space occupied by the fibrous tissue was comprised of hemorrhagic areas. The lobe as a whole was enveloped in a fibrous tissue capsule under which there was some fatty infiltration. The lymphoid half was divided off into lobules, separated by fibrous septa, and the lobules contained each on an average about five corpuscles of Hassal. From this it would seem that

the glandular enlargement was not due to a marked increase in any one element, but that it was naturally large and undergoing the usual changes excepting the hemorrhage."

The upper thoracic aperture in this case was not narrower than normal as was at first thought possible on account of the old



fracture of the clavicle discovered on the radiograph. This fracture must have occurred prior to the child's learning to talk, that is about 2 years before the tracheal symptoms. The upper thoracic skeletal aperture in a child of this age is very small.

Through it must pass the trachea, esophagus, thoracic duct, the innominate, left carotid, left subclavian, internal mammary and superior intercostal arteries; right and left innominate, and inferior thyroid veins; the pneumogastric, sympathetic, phrenic, cardiac and left recurrent nerves; and the longi colli muscles. Besides these, the apices of the lungs covered by the pleura crowd upward as far into the opening as they can get. All these structures leave but little room for the normal thymus gland, and no space for hypertrophy. In addition, the thoracic opening in a given case may be small congenitally. It is manifestly impossible to insert the little finger through this tightly filled aperture without compressing the trachea and asphyxiating the patient. For this work in this case, I found useful the long tracheal canulae that I have used for many years in work on the trachea stenosed from causes other than thymic pressure.

These canulae are made for me in varying lengths and sizes (Fig. 3) for emergencies in tracheo-laryngeal work, but often one especially adapted to the case has to be made, so as to reach the bifurcation without occluding the bronchial orifices. These canulae have saved many lives that must have been lost had the ordinary canulae been used.

As to whether the pressure caused by crowding the finger down through the already full upper thoracic opening caused a laryngeal spasm or not, I am unable to say, but it did not cause any laryngeal paralysis, as an examination the next day showed the glottic movements to be normal.

RESUME.

This is the seventh case on record of the cure of "thymic asthma" by thymectomy, the first case demonstrated radiographically, and the only case where the mechanical pathology of the disease was proven by direct tracheoscopic examination of the living patient.

Thymic tracheo-stenosis is a more accurate term than "thymic asthma" and is acceptable now that the mechanical nature of some cases has been proven. Let "thymic asthma" be applied to cases supposed to be associated with neuropathic, convulsive, lymphatic, rachitic, hemic or other pathology, if desired, until their exact pathologic mechanism shall have been demonstrated, as has been done beyond doubt in the tracheo-stenotic case herewith reported.

No case of thymic tracheo-stenosis can die of asphyxia, if a surgeon be at hand in time with a very long tracheal canula, bronchoscope, or similar tube which will reach below the obstruction. All the cases that have died after tracheotomy have seemed from the reports to have died for want of these long tracheal canulae. These canulae may be worn indefinitely, but a better procedure in thymic cases is to extirpate the thymus gland, partially or totally, or an exopexy may be done. The long tracheal canulae prevent asphyxia from tracheal compression while the finger of the surgeon is passed into the mediastinum to break up adhesions and remove the gland. This removal cannot be done satisfactorily by pulling the gland upward with forceps, as the gland would be torn.

CONCLUSIONS.

1. Friedleben's dictum *es giebt kein asthma thymicum* is an error. The thymus gland did, in this case, compress the trachea sufficiently to diminish and obliterate momentarily its lumen. Thymic tracheo-stenosis seems a better term for this class of cases.

2. The dyspnoea in thymic tracheo-stenosis is worse in the erect position, and it is expiratory, as might be expected from the increased intra-thoracic expiratory pressure, and as demonstrated tracheoscopically in this case. The mechanism of this was demonstrated by the flopping out and in of the elevated gland before it was severed.

3. A radiograph is a valuable diagnostic aid.

4. An absolutely positive diagnosis can be made with the tracheoscope. Upper tracheoscopy is probably not safe in these cases. Tracheotomy should be done under infiltration anesthesia, and should be high, so as to be as far as possible away from the thymectomy wound.

5. A long tracheal canula, reaching to within a centimetre of the bifurcation, renders the breathing free and the operation of thymectomy safe from risk of asphyxia.

6. Thymectomy is indicated, and is best done by the insertion of the little finger from above downward behind the sternum, through a transverse incision after double sterno-cleido-mastoid tenotomy. The insertion of the finger should be of brief duration, as, though the patient with the long canula inserted is safe

from asphyxia, there seems to be serious cardiac inhibition, probably from compression of the nerve trunks about the esophagus. Care should be taken not to injure the pleura.

7. An almost complete thymectomy is without effect upon either the blood or nutrition.

BIBLIOGRAPHY.

SIEGEL. *Berlin. klin. Wchnschr.* Vol. 33, 1896, p. 888.
KOENIG. *Deutsche med. Zig.,* Vol. 18, (27), 1897, p. 274.
EHRHARDT. *Arch. f. klin. Chir.,* Vol. 78, 1905, p. 599.
CARTER. *Med. Rec.,* Apr. 2, 1904, p. 529.
JACOBI. *Trans. Assn. Am. Phys.,* Vol. III., 1888. p. 297.
HEDINGER. *Jahr. f. Kinderh.,* Vol. 43, No. 3. 1906.
THODOR. *Jahr. f. Kindern.,* Vol. 43, No. 5. 1906.
CHKARINE. *Rousski Med. Vcsnik,* Feb. 1, 1904.
PERRUCKER. *Gaz. Hebdom de Med. et de Chir.,* 20 July, '99.

DISCUSSION.

DR. GREENE—These cases of trachea obstruction in young children are exceedingly interesting. It is interesting to compare the symptoms in these cases with cases of tracheo-stenosis. As Dr. Jackson has pointed out, the one marked difference is in the character of the dyspnoea in thymic obstruction. Cases of thymic obstruction seldom reach adult age and we are justified in taking extreme measures. The X-rays, I think, should always be used in diagnosis of these cases of trachea obstruction. It is a most valuable aid, as shown in this photograph by Dr. Jackson.

DR JACKSON—I have not anything further to add except to thank Dr. Greene. I also thank Dr. Mosher for the additional point that I hadn't thought of, and that is an exceedingly valuable point because of the possibility of a case of this kind coming in with a history of a foreign body. It is a case where the ordinary tracheotomy will not relieve the dyspnea. There have been cases reported where tracheotomy has been done and the child has died apparently from tracheal stenosis, because the ordinary tracheotomy canula was not long enough.

PAPER:

AN UNUSUAL CYST OF THE LARYNX.

D. CROSBY GREENE, JR., M.D.

Cysts of the larynx are benign tumors which, when situated on the epiglottis, are ordinarily inoffensive and may attain considerable size without producing serious disturbance. The following case which came under my observation at the Boston Children's Hospital last summer was unusual in point of size and in the resulting obstruction to breathing and swallowing which became a serious menace to the health and even the life of the patient:

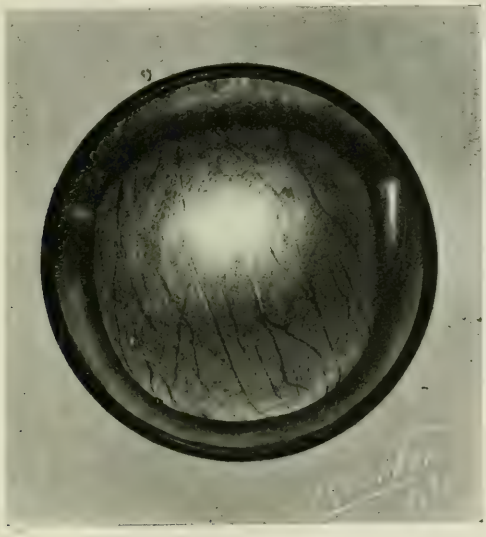
ANNIE C. ———, nine years of age, was admitted to the hospital on July 27, 1906, on account of dyspnoea. The parents gave this history. The child was born prematurely at seven and a half months, but was healthy up to the age of two months. At that time she began to have a cough which had persisted fairly constantly ever since. She had never breathed quietly. For three weeks previous to her admission to the hospital her breathing had been becoming gradually more and more difficult. She had taken very little food and was rapidly losing flesh and strength. When she lay down the difficulty in breathing was increased. She slept poorly and sat up in bed most of the night.

PHYSICAL EXAMINATION. The child was small for her age, pale, emaciated and drowsy. Her breathing was open-mouthed, labored and accompanied by a loud, low-pitched, inspiratory sound, dilatation of the alae nasi and supra and infra-sternal retraction. On examination of the chest a small area of dullness was found at the base of the left lung below the angle of the scapula over which the breathing sounded harsh. External physical examination was otherwise negative.

The nose and upper pharynx presented nothing abnormal. Laryngoscopic examination disclosed a smooth bluish red, globular tumor a little over an inch in diameter involving the right half of

epiglottis and the right pharyngo-epiglottic fold, in contact with the base of the tongue in front and with the posterior wall of the pharynx behind. It pushed the epiglottis well over to the left and completely concealed the cavity of the larynx. It was firm and tense to the touch and not apparently fluctuant.

In view of the marked dyspnoea and the extremely weakened condition of the child it seemed to me advisable to perform tracheotomy before attempting to remove the tumor. Accordingly, on July 28, I did a rapid tracheotomy under light ether anaesthesia, preceded by atropine gr. 1-300 given under the skin. The child



Cyst of epiglottis. Drawing of laryngoscopic image enlarged. Author's case.

made a quick recovery from the operation and immediately began to improve not only in the ease of respiration, but in general condition. She slept comfortably for the first time in weeks, and for the first twenty-four hours after operation slept most of the time. She was still unable to take solid food but took a good quantity of liquid nourishment. Two days after the tracheotomy a change in the appearance of the tumor was noticeable: It appeared a little larger and the surface color had changed from a dusky bluish red to a yellowish hue. Against this background the small red ramifying surface blood vessels could be easily seen. The accompanying drawing was made for me at this time by Mr. Aitken.

On August 7th, eleven days after the tracheotomy, the child having gained much in strength, I attempted to remove the tumor by means of a snare. The child was etherized in Rowe's position through the tracheotomy tube and the cyst seized with tenaculum forceps and drawn into the loop of the snare. At this point the forceps tore out, rupturing the wall of the cyst and emptying the contents, a brown fluid, into the mouth. With the collapse of the

Cyst of epiglottis in two weeks' old infant. Natural size. Case of Dr. Mix.

cyst the parts assumed relatively their normal position, the epiglottis reverting to the median line, and I discontinued the operation. The tracheotomy tube was removed on the following day.

During the succeeding days the tumor began to develop again, and in a week was about one-half its former size, but gave no obstruction to breathing or swallowing. On August 17th, the child was again etherized and I removed a portion of the cyst wall with

biting forceps. This resulted in the evacuation of about a teaspoonful of pus. The cyst had evidently become infected at the first operation with some mild pyogenic organism which did not produce any systemic disturbance, for the child had no fever and gained steadily in general condition while the cyst was refilling.

Microscopic examination of the cyst wall, by Dr. H. C. Low, showed a wall of granulation tissue infiltrated by lymphocytes and polymorphonuclear leucocytes with squamous epithelium on both surfaces, i. e., a subacute inflammatory process in the wall of the retention cyst. There was no evidence of tuberculosis or indication of embryonic tissue cells. There has been no recurrence of the tumor since the last operation. A certain amount of inflammatory swelling remained at its site for a time, but gradually diminished. Laryngoscopic examination showed only slight thickening of the right pharyngo-epiglottic fold. The child is in excellent health.

Through the courtesy of Dr. C. M. Mix of Muncie, Indiana, I am enabled to add the report of another grave case of cyst of the epiglottis which came under his observation. Dr. Mix is preparing a detailed report of this case for publication.

The patient was a newborn infant who had marked inspiratory dyspnoea from its birth. The difficulty in respiration increased up to the end of the second week, when the child died from the obstruction. At autopsy the condition was found to be as shown in the excellent photograph taken by Dr. Mix. This is the third reported case of cyst of the epiglottis in the newborn causing death which I have been able to find. It is possible, as Dr. Mix suggests, that these tumors are of more frequent occurrence in the newborn than has been supposed, and that if the profession were on the lookout for them more cases would be found.

Since 1863, when Gibb[1] first observed and operated on a cyst of the right vocal cord, a large number of cysts of the larynx have been recorded. Beschorner[2] in 1877, in an article reporting a case of cyst of the epiglottis, stated that in a series of 693 cases of tumors of the larynx which he had collected, 45 or about 6 per cent were cysts.

The distribution of cysts of the larynx was found by Moure[3] in 117 cases to be as follows:

Epiglottis	50
Vocal cords	45
Ventricles	8
Arytenoids	4
Ary-epiglottic fold	3
Cartilage of santorini	1
Ventricular bands	2
Site not given	4
	117

Purseigle[4], in a review of all reported cases of tumors of the epiglottis up to 1905, found that 40 per cent were cysts. These cysts are generally of small size, the great majority being not larger than a small pea. A few as large as a hazel nut have been reported. Hamilton, of Montreal, operated on a cyst of the epiglottis as large as a small hen's egg, in a youth of sixteen years, which is, I believe, the largest on record.

Cysts have caused death from asphyxia. Edis[7] and de Cheveney[8] have each reported a case of congenital cyst of the epiglottis in the new born which caused dyspnoea, cyanosis and death in twelve hours. On the other hand, cysts of small size do not give rise to serious symptoms and may be carried for years without special discomfort. They may be spontaneously evacuated without leaving any trace of themselves as in three cases reported by Beshorner[9].

In structure and origin there are three varieties:

(1) By far the most common is that due to obstruction at the mouth of a mucous gland with retention and accumulation of the secretion of the gland.

(2) A second variety has been found on the vocal cords due to accumulation of lymph either in the lymph vessel or in the lymph spaces of a polypoid growth on the cords.[10]

(3) The third variety includes those which are derived from embryonic tissue cells.[11]

Treatment. Cysts have been cured by simple puncture or incision, but, as this method is likely to be followed by recurrence, a preferable method is removal either by means of a snare or by avul-

sion with forceps. Preliminary tracheotomy is only indicated with extreme dyspnoea.

BIBLIOGRAPHY.

1. *Gibb. Arch. of Laryngol.,* Vol. I, p. 24, Mar. 1880.
2. Beschorner. *Berlin. klin. Wchnschr.,* Oct. 15, 1877, Vol. xlii, p. 620.
3. Moure. Etude sur les kystes du larynx, Paris, 1881.
4. Purseigle. These de Lyon, 1905.
5. Schwartz. Tumors du Larynx, p. 87.
6. Hamilton. *Montreal Med. Journ.,* Aug., 1899.
7. Edis. Trans. Obstet. Soc., London, 1877, Vol. xviii, p. 2.
8. P. de Chevenéy. Soc. de obstet. de gynecol. et de pediatri. de Paris, June, 1902.
9. Beshonner.
10. Louys. *Rev. de Chirurgie,* Dec., 1899.
11. *Ibid.*

PAPER:

A CASE OF CHRONIC SINUSITIS, CURED BY INTRA-NASAL TREATMENT.

J. PAYSON CLARK, M. D.

My object in reporting the following case is simply to present a rather striking example of chronic inflammation of the frontal and ethmoid sinuses and the antrum of Highmore cured without recourse to any external cutting operation. There was nothing original or novel about the method of procedure. It was simply a case of perseverance. Had the patient's condition permitted, there is not the slightest question that a radical operation would have been advised, as it was clearly indicated.

It seems to me that reports of cases, such as these, are particularly valuable at this time when enthusiasm is strong over external operations, especially the Killian, and when every 'ologist is so ambitious to add to the number of such operations that he has performed that he may sometimes find it difficult to resist the temptation to do a radical operation on some case which, for one reason or another, were better treated in a more conservative manner.

If any of those here have such a case now under his care, he may derive some encouragement to persist in a conservative course from my experience.

Mrs. J. R. W., aged 64, came to me first in July, 1905. She stated that she had a severe cold about five months before and that there had been a purulent discharge from the left nostril ever since with some frontal pain and tenderness. The form was not so severe as to be unbearable, nor was it constant. The purulent discharge was rather profuse. Examination of the nose showed septum thickened and deviated to the left, causing considerable obstruction of that nostril, which was increased by the swollen turbinates, the middle turbinate remaining in contact with the septum, even after being shrunken with cocaine, while creamy pus bathed both turbinates. The patient's general condition was decidedly poor. She was a small, slight, delicate woman, who had been somewhat of an invalid for a number of years. Her pulse was never less than 100 when she came to my office and she got out of breath very easily. The exertion of merely coming in the cars to see me exhausted her very much. Under those circumstances even the intra-nasal treatment could not be very protracted at each visit, at first. Later, by directing her diet and making her live out of doors as much as possible, her general condition improved considerably.

Her left antrum was seen to be dark on trans-illumination and a puncture with a medium sized trochar was made through the inferior meatus and considerable odorless pus washed out. The antrum was washed six times in the next ten days with a normal salt solution containing about four grains of boric acid; but there was no noticeable improvement. Realizing then that the ethmoid and also the frontal sinus were probably affected the next step was to remove, by cold wire snare, the anterior portion of the left middle turbinate which completely filled the upper portion of the narrow nostril and blocked the drainage from these sinuses. The patient was so much exhausted by her visits (it took her an hour to come from her home to my office) it was deemed best to give only one treatment a week. For the next three months treatment consisted in working out the pus which collected in the nostrils and removing tissue from the ethmoid region with cutting forceps and curettes. In the latter procedure I attempted to work in the direction from which pus appeared to come. By this time I had almost begun to despair of being able to cure the case by intra-nasal treatment. While

the pain and tenderness was less and the patient thought that the discharge was less through the week, the character and amount of pus showed little if any change since the first visit. But the general condition still precluding radical operation under general anaesthesia, there was no course left but to abandon treatment entirely or persist on the same course as heretofore. As I have already partially indicated, this was to cocainize the nose thoroughly by means of pledgets of cotton soaked in an 8 per cent solution of cocaine, and then to further shrink the tissues by 1-1000 solution of adrenalin applied in the same way. The ethmoid region was then carefully wiped out and the source of any pus that was seen was followed up carefully with a probe and the diseased tissue in that region removed by means of cutting forceps and delicate curettes. Guided by the pus in this way I, one day, introduced the probe into the frontal sinus and following the same course with a small sized curette I removed considerable granulation tissue and enlarged the fronto-nasal duct. In using the curette in the duct I was careful to exert pressure forward and downward only. I then washed the frontal sinus with a mild anti-septic solution through a small silver canula introduced through the duct. This treatment was continued for the next three months at intervals of a week or ten days. The antrum was also washed at each visit. There was a gradual improvement during this period. The amount of pus washed from the antrum diminished and became more mucous in character. This was true also of the washings of the frontal sinus. Frontal pain and tenderness had now disappeared. Occasionally after washing out the frontal sinus I injected a 70 per cent solution of alcohol. This was not painful and I believe was of benefit. During the next two months the patient was treated six times. She had come down from using a dozen or more handkerchiefs a day to only one and had little or no discomfort. There was still a moderate muco-purulent nasal discharge and a lump of mucous pus could generally be washed from the nose. About a month later she made her last visit. My notes say, "July 10th, 1906. No pus in nose. Both antra and frontal sinuses transilluminated. The patient has no throat or nose symptoms. A letter, dated December 14th, 1906, in answer to one of inquiry from me, says: 'I am glad to be able to tell you that your cure was *complete*. I have had no trouble from my head or nose, though I have, once or twice, had a cold which affected my throat considerably.'"

The treatment extended over a period of about one year, during which time the patient made forty-five visits.

DISCUSSION.

Dr. Kenefick—I was very much interested in Dr. Clarke's paper, which, I think, is a very timely one at this particular period when elaborate external operations are so readily undertaken. In an experience of more than ten years, I have never had occasion to enter the frontal sinuses externally. We all know that very severe cases of purulent sinusitis of all the sinuses or cavities of the head occur without attracting any special attention on the part of the patient beyond noticing that they had what they describe as an "awful catarrh." This thing characterized as "catarrh" has undobutedly been a purulent discharge. In many cases these is spontaneous healing without any special treatment. Then, in those cases which seek treatment, it seems to me that if they were treated in the acute stage according to these conservative lines, that they ought to be cured. When they get into that condition which is ordinarily said to require an "external" operation, the removal of the middle turbinal or its anterior end should be the first step. That, I have found sufficient in curing ordinary cases of acute antrum or frontal sinus trouble. The question seems to be one of drainage largely, and the natural openings are closed up, by swollen tissue or by chronic redundancy at the point of opening. I think that intra-nasal surgery, such as the removal of the anterior end of the middle turbinal, should be undertaken in any acute case which goes on over a few weeks without recovery. Then, in the chronic cases, I believe in Dr. Clarke's methods, and those are the methods which I have followed in my own practice, and I think that no case should go to external operation until the middle turbinal at least has been removed and an attempt made to provide suitable natural drainage.

Dr. Holmes—Just one word in regard to the so-called radical operation and in regard to the percentage that are absolutely cured, or the percentage that are relieved. I am not antagonizing in the least the radical operation, yet, like most of the men who have spoken, I would not adopt it in cases unless it was indicated at first. I would advise more simple methods. In cases I have seen, of my own and some others, when we felt we had a cure, those cases may show up to us in perhaps a year, or a year and a half, and they may still have some pus or secretion especially in cases where there has

been a great deal of interference and where we have destroyed a great deal of the secreting membrane; and even those cases that have been under the radical operation, are not, in many instances, to our liking.

Dr. Chenery—I would like to say that my last case was a case of double frontal sinus. I had tried all sorts of intra-nasal treatment and had watched it over a year. It was the case of a young lady about twenty-five. She had no relief from her symptoms and besides she was becoming more or less morose on account of her condition. About five weeks ago I operated upon her. I found both frontal sinuses diseased and filled with polypoid degeneration. I don't believe that any intra-nasal treatment would have cured her. I think the operation was necessary. I am only sorry I didn't do it months before.

Dr Clarke—I simply want to thank those who have taken part in the discussion and to bring out certain points which I like to see emphasized. I want to say again, as I stated in my paper, that I should have been the last person to have persevered in this treatment had it not been that the condition of the patient was such that I felt that the operation, under general anesthesia, would have been too dangerous. I put the question to her as regards the general operation, the radical operation, under ether, and she agreed with me that she would not like to take the risk and that is the reason I persevered in the intra-nasal treatment. I simply report the case to show what may be accomplished under such conditions. As regards entering the frontal sinus through the nose, I agree with Dr. Harris, that it is not a safe procedure in a great many cases. In my case, as I said, it was only after a long time that I attempted to pass the probe to the frontal sinus and then only by following out carefully the point from which the pus came.

PAPER:

THREE CASES OF LABYRINTHINE SUPPURATION COMPLICATING PURULENT OTITIS MEDIA.

EDWARD BRADFORD DENCH, M.D.

Suppuration of the labyrinth is a subject which has attracted more and more attention on the part of the otologist during the past decade. Owing to the fact that the labyrinthine fluid is separated from the tympanum only by the foot-plate of the stapes and by the comparatively thin membrane of the round window, it seems rather surprising that invasion of the labyrinth is not more frequently reported as a sequel of chronic middle ear suppuration. Unquestionably, the labyrinth is more frequently involved in a suppurative process than the records of most otologists show. Our attention has been drawn to this complication of purulent otitis media chiefly in connection with fatal cases, and from the fact that, in quite a large proportion of fatal cases—especially in those instances where death has followed as the result of cerebellar abscess—the path of invasion has been traced directly from the middle ear, through the labyrinth, through the internal auditory meatus, to the cerebellum.

Quite frequently, a suppurative inflammation of the labyrinth is found at the time of operation, although, from the clinical history, the condition could not have been diagnosticated prior to the operation. In other instances, however, there is a distinct history, which should lead the operator to suspect labyrinthine involvement.

In the cases about to be reported, such a clinical history was given in one case. In two others, the labyrinthine involvement was found at the time of operation, there being no suspicion on the part of the operator, at the time surgical interference was advised, that such a condition existed.

The first case was that of a young man, of 31, who presented at the New York Eye and Ear Infirmary, with a history of chronic suppurative otitis media, which had lasted for a period of years. Within the two weeks before the patient came under observation,

he had suffered from severe vertigo and headache. The vertiginous symptoms had been steadily increasing. The case was reported to me simply on account of the chronic suppuration, and the consequent indication for relief by a radical operation. After obtaining the history, I expressed the opinion that we should very likely find suppuration of the labyrinth. The radical operation was performed in the usual way. When the horizontal semi-circular canal was exposed, a carious area was found which involved the entire thickness of the canal wall. Curettage easily opened the lumen of the canal. This opening was enlarged until the lumen of the bony canal could be distinctly seen. The carious canal wall was cautiously removed forward to a point just above the oval window. The crurae of the stapes were carious. The stapes was removed, and the entrance to the oval window was enlarged. In this way, free drainage of the vestibule was obtained. A narrow strip of iodoform gauze was inserted into the oval window, and was also made to cover the opening in the horizontal semi-circular canal. The entire radical cavity was then packed with iodoform gauze, and the wound sutured. An uninterrupted recovery took place in this case, and the vertigo entirely disappeared after the operation.

In this instance, invasion of the labyrinth undoubtedly took place through the horizontal semi-circular canal. The involvement was so extensive, that it seemed wise to the operator not to rely upon drainage of the carious area alone, but to secure complete drainage of the labyrinth by removal of the stapes, thus securing drainage of the vestibule. The complete recovery of the patient seemed to justify the operative procedure. Two months after the operation, the acoumeter was heard at 6 inches, in the affected ear, and the forced whisper, at 18 inches. There was still a slight odorless discharge from the canal.

The second case was that of a boy, aged 14, who had been suffering from a chronic suppurative otitis media, involving the right ear, for a number of years. There had been no symptoms indicative of labyrinthine involvement. On performing the ordinary radical operation, an eroded area was found, leading into the horizontal semicircular canal. The outer wall of the bony canal was removed for a considerable distance, until the probe could be passed downward, forward and inward into the vestibule. A narrow strip of gauze was carried into the opening into the semicircular canal, the radical cavity packed with iodoform gauze, and the ordinary

dressing applied. This patient made an absolutely perfect recovery. An examination of the case six weeks after the operation, showed the ear practically dry, there being but a slight serous discharge. The hearing upon the right side, for the acoumeter, was 1 foot, the forced whisper, 3 feet. It should be stated in this connection, that this boy was suffering from a double suppurative otitis media, and that the radical operation was also subsequently performed upon the left side; there was no labyrinthine involvement upon this side. Upon the left side, the acoumeter was heard at 6 feet, and the forced whisper at 9 feet. The tests of hearing made prior to operation were somewhat unsatisfactory, but in this particular case, seemed to indicate that the patient's hearing was considerably impaired, as the result of operation, particularly upon the right side, that is, the side upon which there was labyrinthine involvement.

Two other similar cases, occuring in children—one in an infant only a few months old—have also been operated upon within the past year, by the same method, and in each instance, recovery has been perfect. In the case of the young infant, ten weeks of age, an erosion of the horizontal semicircular canal was found complicating an acute otitis. Here, the radical operation was not performed. A simple mastoid operation was done, and the eroded area on the outer wall of the external semicircular canal was curetted, opening the lumen of the canal. The wound was dressed in the usual way, care being taken to shut off the lumen of the canal from the remainder of the wound, by a strip of iodoform gauze packing. Complete recovery took place in this case. I cite this case simply to show at how early an age we may have erosion of the labyrinthine wall.

In the third case, that of a girl, aged 12, suffering from chronic middle ear suppuration, with no symptoms of labyrinthine involvement, the radical operation was performed simply for the relief of the suppuration. Toward the end of the operation, the region of the oval window was found to be filled with granulation tissue. An attempt was made to remove this, and although the curette was used with the utmost gentleness, the stapes was extracted. The stapes apparently lay loose in the oval window and the foot-plate was considerably atrophied. The proble could be passed directly into the labyrinth. A strip of gauze was packed into the *pelvis ovalis*, and the radical cavity dressed in the usual manner. This patient had rather persistent nausea for forty-eight hours after the opera-

tion, but ultimately made a complete recovery. One month after the operation there was only a very slight amount of discharge from the ear, and the acoumeter was heard at 3 feet on the affected side, the forced whisper at 3 feet.

These cases are reported to show the frequency with which the labyrinth may be involved. The three cases specifically cited occurred during my last twenty-eight radical operations. From the history of these, we find that there are two avenues by which the labyrinth may be invaded: The first and most common, through the bony wall of the horizontal semicircular canal; and second, directly through the oval window. It is a question in my own mind as to whether, in many of the cases where we have suppuration after a complete radical operation, we have not to do with a suppuration of the labyrinth. All who have performed many radical operations, must have been impressed with the fact of how frequently we find granulation tissue in the *pelvis ovalis*. Many of us rather hesitate to interfere with granulation tissue in this neighborhood, for fear of extracting the stapes. I am inclined to believe that we have been rather too conservative in this direction. Granulation tissue about the stapes should be treated the same as granulation tissue in any other part of the tympanic cavity, and if it is necessary to remove the stapes in order to free this portion of the tympanum from granulation tissue, the stapes should be removed. Naturally, if there is no evidence of suppuration of the labyrinth after removal of the stapes, the oval window should not be enlarged, and the operator should content himself by simply shutting off the *pelvis ovalis* by means of a gauze drain, to prevent involvement of the labyrinth through the middle ear. If, however, pus is seen to flow from the oval window, the oval window should be enlarged forward and downward, so as to completely drain the vestibule. In those cases where the horizontal semicircular canal is the site of invasion, the procedure will depend upon the extent of the labyrinthine involvement. If no pus flows from the semicircular canal, after the opening is enlarged, and if no granulations are present about the oval window, the operator may content himself simply by draining the labyrinth through the horizontal semicircular canal, and from the cases which have come under my observation, I can say that he will feel fairly certain of eradicating the labyrinthine involvement by this method. If, however, there seems to be extensive involvement of the labyrinth, the

opening of the semicircular canal alone, will not suffice. After the canal has been thoroughly drained, the stapes should be extracted, provided its foot-plate is in position, and the oval window should be enlarged downward and forward, so as to freely drain the vestibule.

The cases reported are also interesting as showing the effect of interference with the labyrinth, on the power of audition. In all of the cases the hearing was considerably worse, as one would naturally expect. For this reason, therefore, the surgeon should avoid opening the bony labyrinth, unless he is absolutely certain that disease exists in this region; in other words, it should not be opened either through carelessness or for purely experimental purposes, as drainage, even of the semicircular canal will probably cause some impairment in the hearing, owing to the change which it causes in labyrinthine pressure. At least, this seems to have been the result in my own cases.

Regarding the advisability of extracting the stapes, I naturally believe that the stapes should be left *in situ*, in performing the radical operation, unless the *pelvis ovalis* is so filled with granulation tissue that it is impossible to clear this away without removal of the ossicle. In cases where the ossicle comes away during cautious curettement of the oval nitch, we will always find, probably, that the stapes is diseased. The cautious extraction of a healthy stapes should cause practically no interference with audition, as has been proven in many cases where the stapes has been removed either intentionally or accidentally during an ossiculectomy. If granulations are found in the oval window, and upon curettement the stapes comes away, the operator should pass a delicate probe into the *pelvis ovalis*, to determine whether the oval window still remains closed by fibrous tissue, or whether the probe can be introduced directly into the vestibule. Naturally, the utmost gentleness must be used in conducting this manipulation, as otherwise, the membrane of the oval window might be ruptured, and infection of the labyrinth take place at the hands of the operator. If the vestibular membrane is found to be ruptured, I do not think it wise to enlarge the oval window, unless pus can be actually seen coming from the labyrinth. If the removal of the stapes shows a suppuration of the labyrinth, that is, if pus flows from the oval window after the stapes has been removed, enough of the external labyrinthine wall should be removed to afford free drainage. This is best

done by the cautious use of a slender gouge and small curette, enlarging the oval window downward and forward. In other words, removing the promontory and destroying the first turn of the cochlea.

Still freer drainage can be obtained in these cases by removing the external wall of the horizontal simicircular canal as well, and this, I believe, should be done in cases where the labyrinthine involvement is extensive.

When the external semicircular canal and the vestibule are both open, the operator must remember that it will be impossible to break down the bony wall completely between those openings without destroying the facial nerve where it runs above the oval window. There is no necessity, however, for breaking down this small bony wall. The wall of the external canal can be removed cautiously as far as the *aqueductus Fallopii*, the thin bridge of bone, lying just above the oval window, and containing the facial nerve, being allowed to remain. The presence of this ridge does not in the least interfere with drainage.

DISCUSSION.

Dr. John D. Richards—During the past few years I have had nine cases of labyrinthine invasion, five of actual suppuration and four in which there was a necrosis of various parts of the labyrinth. The usual place of infection, in these cases, has been through the horizontal semi-circular canal, and in one instance there was an extension through the first coil of the cochlea; in another instance through the oval window, and another instance there was perforation through the capsule leading down into the vestibule, the perforation being immediately above the semi-circular canal. All these cases were operated upon and in a large portion of them they showed no symptoms whatever, that is, there were no symptoms calling special attention to labyrinthine invasion, but simply that they were victims of chronic suppurative otitis. Very little mention has been made of nystagmus in these cases. In one of the cases, in which the arches of the semi-circular canals, the cochlea and the vestibule were removed and only the ampullary areas of the canals were left, the patient heard all of the Hartmann set of forks and the thirty-five thousand Koenig rod. There is, as a rule, in the cases where the labyrinth is involved, a tendency to fall towards the opposite side from that involved. In regard to the statement made by Dr. Dench as to the removal of the bone extending

from the oval window, downward and forward, I don't think that, in those cases, that we can confine our operative procedure to such definite limits when the vestibule is involved—the cases which I have seen have had the cochlea involved also. The hopping which Dr. Eagleton speaks of I have tried in several cases in which the internal ear has been involved. The patient hops and upon lighting then falls to the opposite side of involvement. In one instance I had an acute suppuration of the labyrinth which followed an acute mastoiditis. An operation was performed and there was found a perforation through the inner wall of the vestibule, around the origin of the internal auditory meatus. An epidural cerebellar abscess was present and the disease had invaded the petrous portion of the temporal bone, requiring an exenteration of the petrous. In regard to avoiding injury to the facial nerve, which is a question we have got to consider in cases in which the internal ear is involved, I would say that the facial nerve is secondary in importance, but we can, as a rule, avoid injury to it by preserving a gutter of bone, or arch for the nerve to run in, extending from the internal auditory meatus down to the lower portion of the facial ridge. In one of these operations a little incident occurred which shows how unexpected difficulties may arise. In removing the external wall of the Fallopian canal a little ring of bone separated cleavage and encompassed the nerve. It was very difficult to get hold of and to separate from the nerve without injury and it made an exceedingly annoying complication. Paralysis of the facial nerve always occurs when the nerve is exposed throughout its circumference. If the nerve is not injured, that is, not cut across, we will, as a rule, get almost complete recovery in about four months. Every case that I have operated upon, in which the entire labyrinth has been removed has shown facial paralysis and every one of them has recovered to a great extent the function of the nerve at the expiration of from four to five months. In no instance was the nerve injured except through exposure.

Dr Richards—In cases which have come on with fair rapidity, vertigo, in my experience, has been present. Cases that have come on gradually are evidenced by the fact of chronic suppuration in the labyrinth, but the invasion has been so insiduous that absolutely no symptoms whatever could be observed except that in some instances there has been slight syntagmus, and in one instance in which the entire labyrinth was ablated, the patient was some weeks later sit-

ting in a room where a piano was being played. He said it caused such dizziness that he fell over, and for some weeks he could not hear music without having vertigo.

Dr. Dench—In reference to Dr. Phillip's remarks, in certainly one-third of the cases there has been no vertigo at all. The experiment of which Dr. Eagleton speaks, I have never tried. With reference to what Dr. Richards says concerning the facial nerve, in cases where there is very extensive involvement, of leaving a small ridge of bone, I make mention, in my paper, I think, of the desirability of preserving the integrity of the facial nerve except in those cases where you have very considerable destruction of the labyrinth. If you practically take away the entire petrous portion of the temporal bone the facial nerve will suffer. In a good proportion of the cases you can drain your labyrinth thoroughly without sacrificing the integrity of the facial nerve.

PAPER:

A DERMA-PERIOSTEAL FLAP IN RADICAL MASTOID.

EDGAR M. HOLMES, M.D.

The purpose of all operations upon the middle ear and mastoid area has been to obtain drainage for pathological secretions, to remove diseased and abnormal tissue, to open for inspection and treatment areas which are normally hidden from view, or to make a more positive diagnosis when some symptom or symptoms were present which might be due to a grave pathological condition in the brain, in the lateral sinus, or jugular vein.

From the time when only those cases in which nature had gone so far as to burrow through the mastoid cells and cortex and produced a retro-auricular infection or abscess, were relieved by a Wild's incision or similar procedure, the progress has been always in the line of a more extensive and thorough operation; and today nearly all aural surgeons are agreed upon one thing and that is to obtain successful results all necrotic and diseased tissue must so far

as possible be removed, all pockets and recesses which may harbor secretions must be avoided. That the best method in any case is that which will thoroughly remove disease, give perfect drainage and favor a rapid convalescence with a final epidermization of all exposed surfaces.

To accomplish this many kinds of skin and periosteal flaps have been devised. The grafting of skin from other localities has also been extensively practised.

It is not my purpose in this paper to describe or criticise all or any particular method of forming flaps in making skin grafts, nor is it seemingly possible to adopt any method which can be successfully used in all cases. What I wish is to describe a method which I adopted five years ago and one which so far as I can learn has not been described as yet.

The usual method of procuring flaps are to in some manner form a tongue of skin from mastoid area and carry it into mastoid wound or to form a flap from the inner portion of the auditory canal nourishing the same by retaining an attachment to the outer portion of canal. I use the inner superior posterior portion of the skin and periosteum of canal and preserve its attachment to the periosteum covering the mastoid bone.

In performing this operation the post-auricular incision differs from that usually made in that the skin only is incised and that somewhat nearer the auricle, care being taken not to injure the periosteum. The anterior skin flap is dissected and carried forward until the posterior and superior portion of the cartilage of the external auditory canal is exposed. An incision is now carried through the posterior and superior wall of the auditory canal and the auricle thus partially detached is carried forward. The anterior and inferior portion of the canal are not injured. With a semi-blunt knife and periosteal elevator, which I have made, an incision is carried through the skin and periosteum of the bony canal from the incision already made beginning at the junction of the lower and middle third of the posterior wall extending to near the annulus, then by an upward course parallel with annulus to junction of superior and anterior wall, then outward to rim of bony canal. From this the periosteum is incised over the linea temporalis as far back as to give ample exposure of bone. An incision is also carried through

periosteum from beginning of auricular incision below backward and downward to posterior portion of mastoid tip.

Considerable care should now be exercised in removing the auricular skin flap, so as not to injure that or its attachment to the post-mastoid periosteal flap. After this has been completed the mastoid portion is easily elevated with the usual instruments and the whole carried back and wrapped in gauze moistened with sterile normal salt solution. We have now a clear field for performing a radical mastoid operation and after all diseased areas and pneumatic cells whether or not diseased have been removed, the bone is carefully smoothed so that there shall be as few uneven areas as possible, for it is important that the periosteal flap when replaced shall be in as perfect adjustment to the bone as possible. We have now to cleanse the wound and we cannot be too thorough. I have found that a normal salt solution has given as good if not better results than any other. When we are sure that we have as far as possible removed everything which might act as a focus for infection, the gauze can be removed from the flap of skin periosteum, which is now carried forward and adjusted. Unless there has been very extensive disease this flap covers nearly all of the exposed bone; the attached portion of skin taken from auditory canal will now cover the area which was the aditus and attic of middle ear.

The next step is to adjust the auricle and in order to prevent the tendency to produce a subsequent occlusion and to give sufficient room for subsequent treatment it seems well to split the posterior portion of the cartilage canal and then with gut suture the upper segment to periosteum over linea temporalis and the lower mastoid segment to that of tip of mastoid. The skin can now be closed and sutured and a small gauze pad so adjusted as to exert gentle but sufficient pressure upon the mastoid to keep the periosteum in contact with the bone below and the skin above.

The auditory canal is now inspected and the skin flap adjusted if necessary, after which a gauze or gauze wrapped rubber tube is inserted to secure drainage from the middle ear.

The operation I have described cannot always be carried out. A marked periosteal inflammation, a disturbance of much of the periosteum, an extensive adhesion following simple mastoid operation or spontaneous rupture, may make it unadvisable to attempt. After

making first incision we can usually decide the question by inspection of the periosteum.

Again it is not always good surgery to close the post-auricular wound, but the skin flap can be preserved even when the post-auricular wound is drained.

I have employed this method in 32 cases and have obtained excellent results in the majority.

There is not space for a report of individual cases at this time, but I will shortly present them in full.

PAPER:

REPORT OF A CASE OF CEREBELLAR ABSCESS. OPERATION. RECOVERY.

JOHN D. RICHARDS, M.D.

The patient, a male aged 25, had for several years a chronic suppurative otitis media of the left ear, accompanied with but slight discharge, never with pressure symptoms.

On July 3rd, 1906, for the relief of the suppuration, a Schwartze-Stacke operation was performed, revealing a necrotic tegmen tympani and posterior antral wall, with the adjacent dura thickened and inflamed.

The patient's progress was satisfactory until three weeks later, when a tooth became ulcerated and the face swollen. General headache developed. The mastoid wound now discharged freely a foul stinking pus; it was thought that the wound had become infected from the aforementioned source.

On August 1st, 1906, he was re-admitted to the hospital, complaining of severe headache and dizziness. He had vomited several times during the few preceding days. The vomiting was not cerebral in character. He showed a tendency to stagger and to fall, opposite to the side involved. He required support. The history noted at this time no aphasia, no optic neuritis, no nystagmus, cere-

bration was clear, respiration 36, pulse 50, temperature 97 4-5 degrees. Bowels constipated, headache present.

He is re-operated upon with the result that a large walled in, epidural abscess immediately above the sinus knee is evacuated. The base of the abscess is formed by necrotic granulations which invest the dura; its pus is foul and stinking. The temporo-sphenoidal lobe is explored at the time with negative results.

August 2nd. The general appearance is better. Headache has largely disappeared. The pulse has increased in rapidity; temperature is slightly above normal. Cerebration is clear, but questionable haziness of both optic papillae is noted.

August 4th. Undoubted papillitis is present in both eyes.

August 5th. The general symptoms became rather suddenly aggravated, and it was at this time that the patient first came under my observation. An examination revealed the following conditions: Slight drowsiness; a tendency when interrogated to become irritable and restless. His answers, though delayed, were accurate. The patient complained of feeling sick and nauseated, and of intense headache over the entire left half of the head. He supported the head with the hand. The face was dusky and moist with perspiration. During the past two days he had lost a noticeable amount of flesh. The temperature had risen to 102 3-5 degrees; pulse ranged from 72 to 140 per minute. He was sensitive to light. His appearance was that of an extremely sick man.

Pupils: Both pupils were well dilated. The left, corresponding to the involved side, being the larger. Both re-acted quickly to accommodation and to light. The consensual reactions were good.

Double optic neuritis was present, the elevation of each papilla being 1-3 mm. There was marked horizontal nsytagmus. The movements were coarse and jerky. These were made more pronounced by directing the eyes to either the extreme right or left lateral position. The oscillations were most marked when the eyes were turned from the involved side. (There was no abducens involvement.) I mention this because when removing certain portions of the labyrinth for suppurative disease, the same phenomenon is present.

When following the moving finger from left to right, the oscillations were from left to right, and vice versa. The sense of smell is not impaired.

The uvula is deviated to the right side, and occupies a position at right angles to the vertical direction. There is a difference in the innervation of the two sides of the palate in favor of the right.

There is complete left facial paralysis, which I am informed developed about one week subsequent to the original operation. The sensation of taste in the anterior two-thirds of the left half of the tongue is gone. Tactile and pain sense are preserved on both sides.

Reflexes: Both patellar reflexes are diminished; the left, corresponding to the diseased side, is practically absent.

Both the biceps and radial jerk on the left side are absent; on the right side both are present.

In response to tickling the sole of the great toe, there is dorsi flexion on the left side, plantar flexion on the right.

There is no clonus on either side.

The tactile recognition of objects is preserved. There is no aphasia, sensory or motor; no areas of anaesthesia. The position sense is good.

There is marked inco-ordination. On asking the patient to touch the tip of the nose with the index finger of left hand, he touches the forehead or the shoulder, and the finger pursues a devious and hesitating course. He is more accurate with right, though inco-ordination here is also very noticeable.

The grip of either hand is good and about equal.

There is no pain upon percussing the skull, and auscultory percussion reveals nothing.

The diagnosis of cerebellar abscess is made.

OPERATION: (NEW YORK EYE AND EAR INFIRMARY.)

Upon exposing the cerebellar dura the tension is found high. There is no bulging of dura. One inch behind the upper third of the sinus there is an area of cerebellum which, as seen through the dura, appears darker than normal, with the veins engorged. Upon palpation this area seems resistant. A small exploratory incision in the dura was made at this point. A clean knife was now passed horizontally inward for an inch and a half, with negative result. It missed the abscess by going a little too high. A second puncture was made near by with similar result.

An exploratory incision was next made at a point internal to the sinus, corresponding to the posterior antral wall. Upon introducing a grooved director directly backward to the depth of about one-half inch there was evacuated thin fetid pus in considerable quanitty. This incision was now enlarged outward to the anterior border of the sigmoid sinus, which was very far forward, leaving little room for manipulation. A second route was made into the abscess cavity by merging the posterior exploratory incisions. Upon passing an encephaloscope into the cavity through the posterior incision, a large quanity of thick foul pus the consistency of condensed cream, welled up into its lumen.

Slate colored necrotic sloughs were evacuated. The walls of the abscess cavity were thin and, protruding as folds into the mouth of the encephaloscope, prevented an accurate view of its interior. Rubber drainage tubes, which were later replaced by perforated bone tubes, were introduced into both openings, the tubes meeting in the central abscess cavity.

Upon the evacuation of the abscess, the pulse became accelerated. No change in respiration was noticed.

On the following day there was marked general improvement. The headache had disappeared. The pupils, though still dilated, were smaller than previously. Inco-ordination was decidedly less. The total aspect of the patient had changed.

On August 9th, four days subsequent to the operation, the following notes were made in regard to the wound:

The mouths of both drainage tubes are filled with thin slate colored pus, which has lost its fetid odor. In the depth of the cavity necrotic disintegrating masses of brain tissue are still present. Along the track of the drainage tubes there are small pink points of granulations appearing, measured from the cerebellar dura to the inner wall of the abscess cavity the depth is two inches.

The pupils are nearly equal. The inco-ordination has disappeared. The left patellar reflex has returned. Nystagmus is present only when the eyes are directed either to the extreme right or left lateral position.

August 20th. The surface of the dura is red and healthy. The dressings are not foul and there is little pus, with no odor. In the depth of the tube there is a small amount of pus; one or two cotton applicators suffice to remove it. On extracting the tube the

drainage tract retains its patency; no pus wells up into its lumen and its sides are covered with thin whitish slate colored pus which when wiped away reveals a finely granular and red appearance of the walls. Drainage is taking place altogether through the posterior fistula.

The pupils are nearly normal in size, the left being slightly the larger. They respond to both accommodation and light. Horizontal nystagmus, though decreased, is still present in the extreme lateral position. A faint vertical nystagmus is noticed.

Co-ordination is excellent. With both index fingers he touches the nose accurately with the eyes closed. The right patellar reflex is about normal, the left a little diminished. A small slough is still to be seen in the depth of the abscess cavity. This slough is attached.

The further history of the case was uneventful. The anterior tube was extruded at the end of the second week, and the anterior opening closed. The posterior opening closed about the end of the third week.

In handling the case the following points were strictly adhered to:

Upon incising the dura a fresh knife was used at each time for exploratory puncture.

No attempt was made to introduce the finger into the brain, or to indulge in unneccessary manipulation.

Time was not sacrificed in trying to thoroughly clear the cavity of pus and necrotic sloughs at the time of operation. Simply, thorough drainage was established through two openings leading into the abscess cavity, and the cavity was allowed to drain itself. No gauze or foreign substance tending to block the flow was introduced into the cavity other than the tubes which retained their patency, securing constant drainage, and requiring infrequent removal. Gauze does not drain.

No attempt at the removal of necrotic sloughs in the depth of the cavity was later made; these were allowed to disintegrate of their own accord, for attempts at removal showed these clots to be attached through small necrotic vessels to the walls of the cavity, which, if thin, renders it liable to be torn, thus opening new avenues of infection into the adjacent encephalon.

The anterior drainage tract, which was the more inaccessible, was allowed to close prior to the posterior tract, and the quantity

of pus discharged through the latter was noticed for an increase at this time.

The encephaloscope was not introduced into the cavity after the anterior opening had closed; its introduction causes an equal displacement of brain tissue or fluid—pus, which if it has no avenue of exit increases the tension of the walls of the cavity. These being thin, the introduction of the instrument may force pus into the encephalon. This was demonstrated at the time of operation. As the encephaloscope was introduced into the cavity through the posterior opening (before the removal of the obturator), pus was forced out through the anterior opening by displacement. This is a serious objection to any instrument founded upon the principle of the encephaloscope, and illustrates the danger of using it when there is only one opening into the abscess cavity, or when there is only a thin wall between the cavity and the ventricle. I believe a bivalve instrument or a pair of thumb forceps to be far safer.

The aural pus showed mixed infection. The pus of the brain abscess, the bacillus coli communis. The abscess had destroyed about the anterior two-fifths of the left lateral cerebellar lobe.

DISCUSSION.

Dr. Dench: I do not think it does any damage to insert the finger into the cavity of the brain abscess, provided the finger is sterile, although, naturally, the intracranial pressure is somewhat increased by the insertion of the finger. This, I believe, constitutes the sole objection. I heartily agree with Dr. Richards in regard to the encephaloscope. The introduction of this instrument into the brain substance, is certainly much more objectionable than is the introduction of the finger. In the last cerebellar abscess operated on, I employed a device presented at the meeting of the American Otological Society last summer, namely, a pair of light retractors. These retractors are used in the following manner: After an abscess has been located by means of a director, one retractor is passed along the director until it enters the abscess cavity. The director is then removed, and the second retractor is slipped along the first until the abscess cavity is entered. By separating the retractors gently a free evacuation of pus is obtained, and by means of reflected light, the entire interior of the abscess cavity can be inspected. This plan certainly possesses many ad-

vantages over the inspection of an abscess cavity by means of the encephaloscope.

In regard to the mortality of cerebellar abscess, I would say that Dr. Perkins, one of my assistants, has gone over the literature of cerebellar abscess very thoroughly, and I have tabulated reports of 102 cases. These cases will be embodied in a paper which I shall present at some future meeting. Out of these 102 cases, 34 were cured.

With reference to the occurrence of optic neuritis, this symptom was present in 34 cases, it was absent in 37, and its presence or absence, in 31 cases, is not stated.

Regarding the manner in which these abscesses should be evacuated, I would say that the opening of the abscess in front of the lateral sinus was advocated by French surgeons quite a number of years ago. Of the cases reported, in 45, the opening was made behind the lateral sinus. Of these, 25 were cured and 20 died. In two of the cured cases, double drainage was employed—that is, one opening was made behind the sinus and another in front of it. Eleven cases were operated upon by an opening in front of the sinus, and of these, four were cured and seven died. In the remaining 46 cases the method of operation is not stated. Out of this number, 4 were cured and 42 died.

I am inclined to believe that wherever the double opening can be made, that is, where drainage can be employed by making one opening in front of the sinus and one behind it, this method should be the procedure of election.

I hope Dr. Richards does not think that I put the finger in the brain before I make the incision.

Dr. Kenefick: I was very much interested in Dr. Richards' case, having had the privilege of following similar cases operated on by Dr. Dench, in his clinic. Dr. MacEwen has had better success than we in treating these abscess cavities by gauze drainage. I think he uses plain sterilized gauze powdered with boracic acid. It seems that this method in this country, and in our experience at least, has not given the results that he succeeded in getting. I think that Dr. Richards' success in this case is largely due to the "fore and aft" drainage and the fact that he used the tube.

Dr. Bryant: I have been fortunate to see the case which has been so ably described by Dr. Richards. I think he has touched the key-note of success in cranial surgery when he says it is best

not to put anything into the cranial cavity. In the cases I have had 50 per cent have recovered.

Dr. Richards: I should have mentioned that in these cases the question of anaesthesia is very important, and I believe chloroform should be the anaesthetic used. I have seen two cases of cerebellar abscess given ether, and both succumbed to respiratory failure—probably due to oedema of important brain centers to which ether predisposes. In regard to Dr. Dench's statement about introducing a finger into the brain, I don't see the necessity of that. If the finger is introduced for exploratory purposes we are apt to break down the brain tissue and then mistake the broken down tissue for pus. I have seen this occur repeatedly. If I understood Dr. Kenefick correctly, he says that Dr. MacEwen uses gauze for drainage in these cases. I was not under that impression, as some years ago I heard Dr. MacEwen refer to the subject and he used at that time bone tubes for draining these abscess cavities. In regard to the large dural incision spoken of by Dr. Dench, I may say I have seen it practiced a number of times and almost always cerebral hernia has resulted. In these brain cases we cannot, as a rule, localize the abscesses, and what we actually do is to explore the area which is most probably involved, making the diagnosis with the knife. We do not locate the abscess upon the symptoms, nor does the neurologist locate it for us. To make a large dural incision for exploratory purposes and then to have to make another is certainly exposing the patient to very great dangers. I have seen several die purely through cerebral hernia from this cause. I think it is far safer to make a greater number of small incisions, and then, having found the abscess, to make the incision as large as is necessary to meet the indications in the individual case. Putting the finger into the brain disintegrates the tissue, and it cannot be done without disintegrating the tissue, even though the finger is passed through the exploratory incision.

PAPER:

REPORT OF THREE INTERESTING CASES.

T. J. HARRIS, M.D.

Case I:—Papilloma of the Nose.

From the time of Hopmann's celebrated paper in 1883, the subject of nasal papilloma has been a fruitful source of discussion, especially from the pathologic standpoint. Dr. Jonathan Wright, a former member of this society, has done much toward the clearing up of this mooted question. Today it is generally recognized that what Hopmann described as papilloma was not a true papilloma at all but, as Wright calls it, a papillary hypertrophy. True papilloma of the nose is an exceedingly rare tumor. Wright, in his paper on the subject in 1891, while not pretending to exhaust the literature, and frankly admitting the impossibility of running down all the cases on account of the faulty nomenclature and the lack of a microscopic examination, was able to collect less than a dozen. Bosworth had seen one papilloma in 200 benign growths. We have not attempted to search the literature since 1891, but we do not recall seeing any case reported in recent years. On account of the rarity of the affection, as well as because of several features not mentioned by writers on the subject, we desire to report the following case:

Dr. V., age 68, consulted me last spring complaining of difficulty in breathing through the left nostril. He had always enjoyed good health but felt that the difficulty in breathing, together with the habit he had acquired of using cocaine to get relief, was pulling him down. There was no pain in the nose and he had not had nasal bleeding. Examination of the throat and naso-pharynx was negative. Examination of the nose showed a papillary growth obstructing the left nostril and situated about 2 inches inside the vestibule. The probe discovered that this was attached to the septum, to the floor of the nose and to the anterior portion of the inferior turbinal. Inspection of the right nostril revealed a small

amount of a similar growth attached to the septum anteriorly. The growths were removed by the cold wire snare and curette and the bases carefully touched with the galvano-cautery. The microscope showed them to be true papillomata. The patient was seen during the month of December of last year. A slight recurrence had taken place in the left nostril on the septum alone. The patient was feeling in good general health.

A mark of distinction between the true papilloma and the papillary hypertrophy is the situation of the growth. The former is found in the anterior part of the nose while the latter is confined to the posterior portion. With this rule our tumor corresponds. We have no record of a case where the growth was found in both nostrils at the same time. The possibility of a malignant tendency is mentioned by several authorities. In our case, this was considered on account of the age. There was, however, no sugestion of it. That it can occur is shown by the remarkable case of Verneuil in which the papilloma recurred in the larynx and bladder after removal, and death finally resulted. Bosworth says, that they are found in early adult life and more commonly in children. This does not apply in the case just reported, and, on account of the haze which has hung over the diagnosis till recently, we question how much weight can be placed upon this observation. Our patient resembles the others reported in presenting no characteristic symptoms. The development of the tumor is usually a slow one and only an increasing discomfort in breathing is complained of.

Case II—A case of Hyperosmia.

Disorders of the sense of smell are not at all uncommon, especially when they concern an impairment or abolition of that function, commonly known as Anosmia. A great deal has been written upon both the pathology and the treatment of this affection which is generally regarded as an organic disease. Much less frequently met with are the pure functional neuroses involving the sense of smell. Of these Parosmia, or the perversion of smell, is occasionally seen. Dr. Munger, a fellow-member, read a paper on this subject a few years ago, if we are not mistaken, in which he gave some remarkable instances of such perversion. Hyperosmia, or hyper-sensitiveness of the sense, is an exceedingly rare disorder, at least as encountered by the rhinologist. In twenty years of professional work we do not recall seeing but this one case which we

are about to report. Wherever mention is made of this disorder, in text-books upon the nose, hysteria is given as a cause. This may explain why we see so few of these cases, i. e., that they are treated by the neurologist.

Mr. K., age 57, banker, consulted me in November of last year, giving the history that he had been a great sufferer for years because of his extreme averson to odors. On inquiry it was learned that it was only certain odors which produced this distress, especially strong cheap perfumes, cabbage and turnips when cooking and similar substances. These affected him so unpleasantly as to produce headache and nausea with vomiting at times. Further questioning brought out a history of which several attacks of neurasthenia, as well as a chronic stomach difficulty, for he had consulted Prof. Van Norden during the preceding summer. He further stated that in the past, when his stomach had been giving him especial trouble, he had suffered more from the nasal disorder. Examination of the nose showed a moderate degree of chronic rhinitis in the terminal stage. There was no complaint of nasal discharge or stenosis. No contacts of the turbinals with the septum were detected. A diagnosis of hyperaemia, secondary to disturbance of the gastro-intestinal canal, was made. Under gentle local applications of argyrol, 25 per cent, combined with the use of the galvanic current direct to the nose, there has been a most satisfactory improvement in the condition complained of. He has been warned, however, that any renewal of his stomach difficulty or another break-down will bring on the sensory disease again.

The pathology of this and allied functional disorders of the sense of smell offers a most interesting field for investigation. A clinical report, as this alone pretends to be, is not the place for a consideration of this question. We would only remark that while they have been generally ascribed to hysteria, it is greatly to be doubted if that is the usual cause. Certainly this case clearly shows a gastro-intestinal origin for the complaint. What the true connection between the two is, whether it is in reality a reflex, as is generally stated, or whether it is a form of toxaemia, expressing itself upon the olfactory nerve, we are not prepared to say. We only know that the extent of auto-intestinal toxaemia has not yet been fully appreciated.

Case III—A case of Panotitis, complicated by Empyaema of the Nasal Accessory Sinuses, with several unusual features.

We desire to frankly state at the outset, that we are not at all sure that this is the proper title for the case which we are about to report. No case in years has proved more puzzling and, in many particulars, it is still not clear to us today.

Mrs. H., age 48, consulted us on August 28th, 1905, giving the following history: She had always been well; never had had any trouble with the ears till the present attack. She had had several operations upon the nose some years before. The difficulty with the ears began upon December 17th of the preceding year. (You will note that a definite date is given for the onset.) On returning from a small social gathering, she was seized with pain in both ears with intense vertigo and tinnitus. We have no record that she lost consciousness. The ears soon began to discharge. How soon after the beginning of the attack we do not know.

These symptoms, except the pain, have persisted in a greater or less degree till now. She complained particularly of a constant pressure in the back of the head. Treatment at the hands of several competent physicians by the usual methods of inflation and massage had given no relief.

Examination showed a well nourished woman of fifty who was so deaf that she heard only loud conversation in front of the left ear. There was entire loss of the right membrana tympani; the canal was perfectly free from all secretion. The left membrana tympani showed two good sized perforations, one in the center and one in the posterior upper quadrant. There was a slight amount of moisture detected by the applicator. No necrosis was discovered by the probe. The C tuning fork was heard 7-15 seconds in the left ear, which easily perceived the C fork by aerial conduction. An empyaema of the ethmoid cells and of the sphenoid sinuses in both nostrils was recognized, with polyp formation. A series of intra-nasal operations was performed which entirely relieved the empyaema. At the completion of the opening of the sphenoid on the right side, the patient said I had struck the site of her pressure and dizziness. This was indeed greatly lessened by the operation. Gentle cleansing of the left ear caused the slight discharge to stop. There was a moderate improvement in the hearing in the left ear. On October 7th, my record gave the tuning fork-reactions as follows: Left, C_1 3-25, C_2 3-7, C_3 3-7, C_4 2-3. Right, C_1 0-20, C_2 11-10, C_3 0-2, C_4 0-0. Galton was nearly

normal in the left ear at 6 inches. At this time the patient was put upon a saturated solution of K.I., ninety drops a day. Under the use of this there was a most gratifying improvement in the hearing. The vertigo also was decidedly better. On December 29th, I made a very gentle application of vibro-massage to the left side of the head. This I have used in many cases without the slightest unpleasant results. Here it had the effect of bringing back the vertigo in a severe form which lasted for a number of weeks. On May 10th of the past year, she was hearing exceedingly well and only complained of a slight return of the pressure in the head. At this time I made my great mistake. Instead of being satisfied with the marked improvement which had been made, I yielded to her importunities, and, seeking to employ what would be the most harmless treatment I could think of that might do good, I dropped a few drops of a 1-5000 solution of adrenalin chloride in the canal of the left ear. At once the patient was seized with intense pain in the ear. In spite of all I could do, this pain persisted for a number of days. With the pain, vertigo returned, the ear began to discharge and she became almost stone deaf. This deafness has continued ever since. No particular reaction could be seen in the ear nor did the Eustachian tube show an inflammatory swelling, though at the first the pain was referred to that region. Later, the pain was in the bone behind the ear and a mastoiditis was suspected. The Eustachian tube, it might be said, had been carefully studied. It was freely open at all times but, to make doubly sure, on a number of occasions we easily introduced a large bougie without benefit. We desire to add that this solution of adrenalin had been used before, in a number of cases, without the slightest ill effect and was employed after this with equally innocuous results. The solution was perfectly clear in color. It will be noted, too, that the pain followed instantly.

We have called this case one of panotitis. The sudden onset, the profound deafness, the vertigo and tinnitus, together with the results obtained by the functional tests, all point conclusively to an involvement of both the middle and internal ear. Panotitis, as described by Politzer who first used the term, is the result of some severe infection such as scarlet fever or diphtheria. There is always a sudden loss of consciousness. This did not take place in our case. At least we have no record of it. As some eight months had elapsed when we saw her, this may have occurred for a short

space of time, but no mention of it was made in the history given. What was the nature of the invasion in this case? It is perfectly possible that some virus could have entered the ear through the blood or lymphatic system. There was, however, not the slightest indication in the history of any acute involvement of the nose or throat. Granted that this was the case, we feel strongly inclined to regard the long standing disease in the accessory sinuses of the nose as primarily at the bottom of the aural invasion. Whether the middle or the inner ear was the first to be attacked, it is impossible to say. As Politzer states, the onset may be so abrupt that the two are attacked almost simultaneously. The relief obtained to the occipital pressure by the intra-nasal operative work is very suggestive of the close nasal association of the affection. Most incomprehensible of all, is the violent return of all the symptoms by the local use of the adrenalin. We have since learned of a somewhat similar effect of the drug in another case. We have tried to explain it on the ground of a personal idiosyncrasy, and yet this is not perfectly satisfactory. We have wondered if there could have been defect in the inner wall of the middle ear and that the drops had penetrated directly into the inner ear. We had on previous occasions employed a number of astringent and aseptic solutions in this ear without causing the least discomfort, and had used adrenalin freely in the nose on many occasions.

MEETING OF MIDDLE SECTION HELD IN CLEVELAND, OHIO, FEBRUARY 22, 1907.

PAPER:

UPPER TRACHEOSCOPY AND BRONCHOSCOPY.

CHEVALIER JACKSON, M. D.

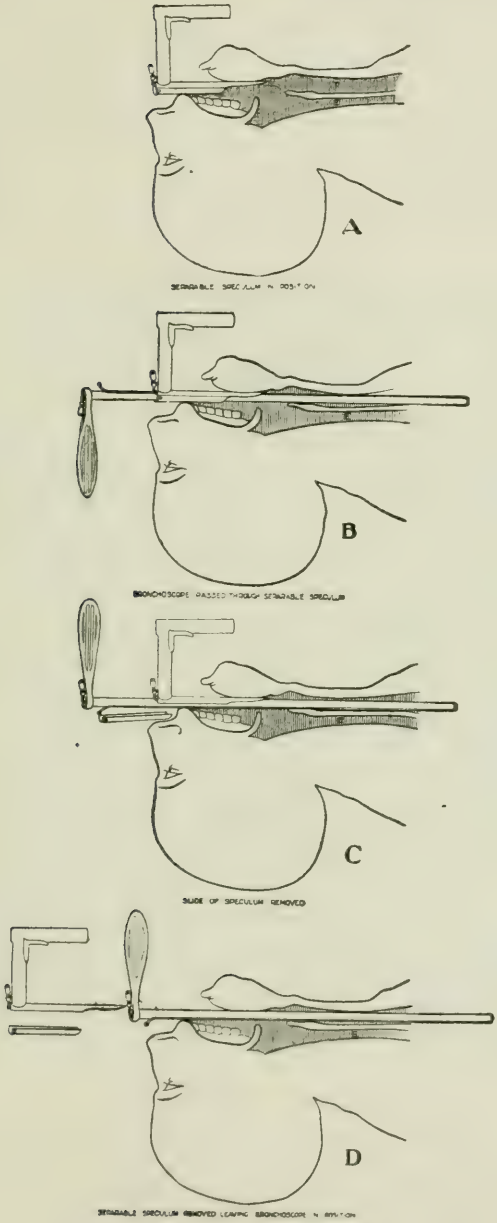
There are a number of ways in which upper bronchoscopy and tracheoscopy may be performed, but the simplest method is as follows:

The chloroformed patient is placed in the dorsal position with the head hanging over the edge of the table, the head being supported by an assistant in the position shown in Fig. 1.

A double bronchoscopic battery is used, one cord being attached to the separable speculum and the other to the bronchoscope. The bronchoscope is held in readiness by an assistant, while the operator slides the separable speculum down over the dorsum of the tongue until the epiglottis comes into view. The epiglottis and the upper portion of the larynx may be painted over with a twenty per cent cocain solution if it is deemed necessary, which it rarely is, unless for some reason deep chloroform narcosis is contraindicated. The separable speculum is then introduced as before, and the epiglottis found. Then the epiglottis and all the tissues attached to the hyoid bone are lifted strongly with the *point* of the separable speculum. It is very necessary to lift with the point and not to use the upper teeth as a fulcrum.

When the separable speculum is in the position shown at A in Fig 2, the axis of the speculum corresponds to that of the trachea, the depths of which are seen below the vocal cords.

The bronchoscope is then introduced through the separable speculum until the tube mouth is near, but does not touch the cords. The eye is transferred to the bronchoscope and the respiratory movements of the cords are watched. When on the inspiratory excursion the bronchoscope is pushed through into the position



SCHEMA ILLUSTRATING UPPER TRACHEO-BRONCHOSCOPY

shown in B. The gag is then inserted and the separable speculum is removed, by slipping off the slide as shown at C, leaving the bronchoscope in the trachea as shown at D.

This is by far the easiest and simplest method of introducing the tube in upper bronchoscopy. With a deeply anesthetized patient it requires in a favorable case but a minute or less to insert the bronchoscope. In children it is equally easy under local anesthesia. In some cases, especially those who have not normal larynges, or in

whom the neck is made rigid by adhesions following the wearing of a tracheotomy canula, it may be difficult. In cases of bilateral abductor paralysis, the cords will not separate and it is necessary to push the bronchoscope through. In such cases it is sometimes easier to pass the bronchoscope with the obturator in place as this prevents abrasion of the mucosa or the hooking of the tube mouth over one or both arytenoids. The proceedure is the same except that the

eye is not transferred to the bronchoscope until the instrument is in the trachea and the obturator is removed. No undue haste is necessary in removing the obturator as it is so made that the air may pass freely.

The latter method with the mandrin has been found easiest by some of the men I have taught, and is used by them for all cases.

The author then exhibited with the lantern a number of endoscopic views of the trachea and bronchi.

DISCUSSION.

DR. COAKLEY: Dr. Jackson's paper and the illustrations and instructions which he has shown are enough to repay one for coming to this meeting. The practice in the use of the instruments on a phantom or the cadaver, will make considerably easier their use on the living subject. I had the privilege years ago of studying this method of examination at the fountain head with Prof. Killian. I was surprised to see the ease with which Killian examined the trachea. They frequently used the bronchoscope in cases of enlarged thyroid glands to determine the amount of pressure on the trachea. I have used this method of examination from time to time. It is the easiest and best way of operating on papilloma of the larynx in children, and I have had an instrument made for such cases. I wish to thank Dr. Jackson for his presentation of the subject.

DR. CANFIELD: I looked forward to hearing this paper with considerable interest. I am sure it would have helped me in a case of extensive edema of the larynx and trachea which I saw several years ago. The patient had swallowed a plate of false teeth which lodged in the oesophagus and then the edema developed. The trachea was almost occluded. A tracheotomy tube was inserted and the thyroid was split. The edema extended so far down into the trachea that I was afraid a more extensive operation would aggravate the condition and I sewed up the wound. I removed the teeth from the oesophagus through the mouth.

PAPER:

REPORT OF CASE OF TRAUMATIC ETHMOIDITIS. CEREBRAL ABSCESS. DEATH DURING OPERATION. AUTOPSY.

J. A. STUCKY, M.D.

The following unique case is presented for your consideration, believing it to be one of a class of cases in which the accessory sinuses of the nose are involved, that calls for more than a passing notice.

A. F. aet. 16—Living in the mountains of Kentucky was admitted to the Good Samaritan Hospital on August 13, 1906, having fallen from a fence four feet high, four months previously, striking her nose on a piece of timber, fracturing the nasal bones—pieces of bone coming out of anterior nares two weeks later. Complains of constant pains through head, more severe at times, especially when stooping or jarred. Pain through eye, especially left one, which is almost blind. Convergent strabismus of the left eye: V. R. 20-20, V. L. 20-80. Pain on deep pressure over left eye, particularly over temples. General health below par, Much cough. Unable to use opthalmoscope on account of irritability, fighting and etc. Family history good. Transillumination of frontal sinus positive. Antrum negative. Vertigo when stooping, pain and dizziness increased by blowing nose. Bridge of nose swollen and tender. Every evidence of fracture. Examination of anterior nares showed cartilaginous septum torn from the vomer, pushed over against the middle turbinate. The middle turbinate adherent to antral wall, and pus oozing above and below tip. Posterior nares full of pus. Probe shows roughness and degeneration of ethmoid cells. On Aug. 15, under general anaesthetic, the septum was straightened, the middle turbinate removed, granulation and pus removed, anterior ethmoid cells and sphenoid sinus curetted. Four days later, strabismus relieved, vision normal, complains of no headache. Did not see patient again for a week. Sept. 4th. Pain increasing for past few days. Nose swollen, discharge ceased. Vision R. & L.—20-15+

1.25, equalling 20-20+. Opthalmoscope shows oedematous retina and papillary neuritis. Is irritable, despondent, threatens suicide. Complains of constant and increasing pains, relieved by cocaine and adrenalin sprayed into the nose.

Sept. 5th. Complains of more pain. Pulse 64, temp. 98. No appetite, restless and very irritable, hysterical—laughing, moaning, crying, alternately. Resp. shallow. Dr. G. P. Sprague called in consultation. Decided to wait 24 hours, and explore accessory sinuses, 1-12 gr. heroin given hypodermatically.

Sept. 6th. Had a good night, is brighter, less irritable and despondent, and anxious to have operation. Is prepared for operation by free purgation, followed by bath and enema. At one o'clock, nurse reports that she has another hysterical attack. At 2:45 I saw her; respiration jerky and shallow, tossing head and throwing arms; would reply to no questions. Pupils still partly dilated but reacted equally to light (had homatropine 24 hours before for refraction) forehead, and upper part of face slightly oedematous and cyanotic. General condition bad, but symptoms of pressure increasing, patient was taken to operating room. Condition improved under ether, requiring unusually small amount for narcosis. Post-nasal tampon inserted. Nasal cavity and ethmoid and sphenoid examined. Granulation tissue and slough, removed with curette. Very little bleeding from nose. Incision for Killian's radical operation on frontal sinus was made; the oedematous tissue bled freely, and was controlled by retractors. Periosteum was very dark color, much thickened, and very soft when removed. Continuous and rapid bleeding from bone for a space around naso-frontal articulation size of a silver dime. At this point the outer table of frontal sinus, which was unusually soft, thin and vascular, was removed with a chisel, and the entire sinus, which was of exceptionally large size was filled with firm, organized clot, which was disintegrating in spots. When the clot was removed, the respiration suddenly ceased, and notwithstanding all the known methods of restoration were used by Drs. Estill, Sprague, Griggsby and myself, the patient never rallied. Death was practically instantaneous. Artificial respiration was continued for nearly two hours. Normal saline solution injected intravenously, strychnia and atropine given hypodermatically.

Notes of Dr. G. P. Sprague—Neurologist. "Patient Amanda F. Sept. 5th, was seen at Good Samaritan Hospital. Is a large well

developed white girl, aet. 16, of fair nutrition, symmetrical build, and intelligent appearance. Is anaemic, pulse varies from 80 to 85, of fair volume and regular. Respiration normal, temperature 97, pupils equally dilated by homatropine; tongue protruded without deviation or tremor. Both knee jerks absent, even when re-inforced. No ankle-clonus, nor Babinsky reflex. Stands fairly well with feet together and eyes shut. Complains all the time in a whining, singsong groan, of pain in the left half of the forehead, from the midline to the ear, and has not had pain anywhere except over the entire frontal bone, where the pain is sometimes worse on the right than on the left. Nose is swelled but not sore. Pressure on R. frontal region causes pain in L. temporal region. Talks quietly for a minute. Laughing and joking with the nurse, calling to nurses whose step she recognizes about the ward, and then suddenly groans and whines for ice, to have her head rubbed, to be fanned and etc. Pettishly refuses to write her name, or anything else, though admitting that she can write. Says she wont if she is asked all day and that she never does anything when she says she wouldn't. Has no symptom of either motor or sensory disturbance, except as noted. Diagnosis: Cerebral abscess or meningitis, caused by inflammation and pus in the frontal and ethmoidal sinus, leaning to the latter.

Autopsy twenty hours after death. Only the brain was examined. There was no fluid in the ventricles, no apparent disease of the dura or the pia arachnoid, except over the left cribriform plate and in the sella tursica, where the pia and dura were both adherent. The left cribriform plate being necrotic, with a fracture through the inner table of the left frontal sinus. The entire anterior and middle cerebral convolutions softened and infiltrated by pale green pus of creamy consistency. Just internal to the cribriform plate in the anterior frontal convolution, there was a thick, tough, gritty substance, the remains, apparently, of an abscess cyst wall, holding approximately one dram, while there must have been several ounces of pus, altogether. The abscess had evidently ruptured into the brain substance, but there was no indication that it had been connected with any sinus or other cavity.

The interesting features of this case are: (1). The extreme rarity of an injury to the nose resulting in infection of the ethmoid cells and frontal sinus. (2). The mental symptoms caused by the resulting septic condition. (3). The strabismus and amblyopia caused by pressure in sphenoidal sinus, which was relieved as soon

as the pressure was released. (4). The absence of focalising symptoms, even when so large an area of the brain is involved in so decided pathological condition. Query—Given a similar case, what would be the best operative procedure? Would I have been justified in searching for cerebral abscess with no more focalising symptoms than presented by this case? It is greatly desired to have the counsel of the Neurologist in cases like this, and others in which we have marked Mental Symptoms accompanying suppuration of the nasal accessory sinuses.

PAPER:

INDICATION FOR RADICAL OPERATION ON THE NASAL ACCESSORY SINUSES.

NORVAL H. PIERCE, M. D.

The following paper has been inspired by a case of pansinusitis which terminated fatally after operation by a fellow practitioner and by the growing tendency on the part of some operators to too hastily deal radically with cases which in the opinion of the writer might be better treated by more conservative methods.

The remarks which follow must of necessity be general in character, as the time allowed for the reading of the papers of this program must be brief.

First, let us consider the question as to the damage to life of suppuration within the accessory sinuses without operation. Pit in his well known report of 9,000 autopsies held at Guys' Hospital reports but 1 case of death attributable to nasal suppuration. Zarinko, quoting Treitel, gives 1 extra dural abscess and 2 cerebral abscesses as of rhinal origin out of 6,000 autopsies at the Berlin Pathological Institute.

Wertheim out of 10,396 autopsies held at the Pathological Institute of Breslau gives only 6 as of rhinologic origin.

Dreyfus, in his Monograph published in 1896 collected fifty-eight cases of cerebral complications from literature of which 5 were

referable to the maxillary sinus, 22 to the frontal sinus, 18 to the ethmoid cells, and 13 to the sphenoid. That is 40 of the 58 involved the fronto-ethmoid cells—the group which is by far the most frequently the starting point of grave complications.

Givens in 1900 collected 142 cerebral abscesses of which only 6 were of nasal origin.

From a consideration of the literature, therefore, we are justified in the conclusion that there is but very slight danger of encephalic complications arising in the course of either an acute or chronic sinusitis.

In contrast to this showing of cases not operated on is the rapidly swelling list of deaths following operative intervention. This phase of the subject has been recently developed in an excellent article by Dr. A. Carpait.

In 1904 Lermoyez, at the French Laryngological Society, remarked that the frontal sinus is rarely a source of danger to life so long as it is not operated on. Luc admitted that a sixth of his cases operated on by the Ogston-Luc method suffered from intracranial complications. He has therefore discarded this type of operation in favor of the Killian method. Killian's statistics give much happier results, but Copart knows of two fatalities following operation by the Killian method, and Thiel reports 1 death in 6 operations.

I will not detain you with exhaustive details of reported cases, but will simply boil the matter down to the statement that experience teaches us at the present time that the mortality of accessory sinus suppuration has been greatly increased by operation interference. The reason for this is imperfect technique, improper selection of cases, and increasing frequency of operating, since the introduction of external methods.

Naturally a clinical parallel has been drawn by the medical mind between the mastoid cells and the nasal accessory sinuses. We are now in a position to realize that this has led to erroneous conclusions. Suppuration within the mastoid is always fraught with danger on account of the immediate proximity of the sigmoid sinus, and the liability of the suppurative process to break down the membranous guards of the two fenestræ with subsequent involvement of the labyrinth and extension to the cerebral contents, while thrombophlebitis and general sepsis from accessory sinus disease can occur only by extension of suppuration through intervening tissue to more

or less remote venous channels. In operations on the mastoid the field is completely accessible to sight and touch. The same can scarcely be said to be always the case with the accessory sinuses. Lastly operations on the mastoid are not followed by that peculiar form of osteo-myelitis which is apt to follow operation on the accessory sinuses, especially of the fronto-ethmoid group, and which depends probably on a thrombo-phlebitis of the diplœtic veins.

As with the mastoid so with the accessory nasal sinuses we may operate either to prevent dangerous complications, to bring suppuration to a close, to relieve disagreeable or unbearable symptoms, or to save life after the dangerous complications have arisen. But we must remember always that operations on the accessory sinuses are fraught with peculiar danger—dangers not met with in mastoid work. He will arrive at the best results who will most prudently weigh the dangers of operation as against the dangers to which the diseased sinuses expose the patient, or as against the relief of symptoms more or less annoying, unpleasant or unbearable.

In a broad way, therefore, I believe it to be a mistake to immediately resort to radical external operations merely because the Skiagraph or intra-nasal examination discloses pus in the antrum, fronto-ethmoidal or sphenoid cells. Satisfactory results may usually be obtained by local intra-nasal procedures. Especially is this true of acute cases; and in chronic cases the external operation should be restricted to those cases which have resisted intra-nasal procedures and in which acute exacerbations show a progressive disease, or when the contributing symptoms are such as to fully warrant us in assuming the dangers inseparably connected with radical operations on the sinuses.

DISCUSSION.

Dr. Vail: The eye symptoms should be observed carefully in making the diagnosis in such cases. In regard to Dr. Canfield's paper, I should think some of the other methods of operation are preferable as they can be done in much less time and accomplish the same results. I have operated a number of times, making a large opening into the antrum through the inferior meatus.

Dr. J. J. Kyle: In reference to Dr. Stucky's paper: The vertigo, halucination and eye symptoms would indicate a brain abscess. In regard to Dr. Beck's paper, atrophic rhinitis may result from chronic suppuration in one sinus and the thorough curetting of such a sinus may relieve the symptoms entirely. The treatment with

the nitrate of silver seems to offer some hope of material benefit, at least in selected cases, particularly where the case is seen early.

Dr. Thompson: I am well impressed by Dr. Pierce's paper. So much radical work is being done that such papers as this are needed. There are a large number of men who take short, post-graduate courses and then do all kinds of radical sinus operations, and it is remarkable that so few meningeal infections follow these operations.

Dr. Richardson: I approve of the suggestions made by Dr. Pierce. It is wise in all cases of sinus infection and especially in frontal and ethmodial infection to weigh well the necessity for an external operation.

Dr. D. B. Kyle: I agree with Dr. Pierce that as physicians, we should be more conservative. My experience has justified me in following conservative lines of treatment. Some men no longer regard the nose as an organ of physiological importance; they consider it simply a hole in the face.

In atrophic rhinitis, one of the important factors in producing the conditions, is a deficient nerve and blood supply, and the structures thus fail to develop properly.

Dr. Roe: The ethmoidal cells should be considered as a possible factor in cases of atrophic rhinitis. The careful study of the individual cells is difficult, but they should be examined and probed and operated upon when necessary. In regard to radical operations of the accessory sinuses, I believe with Dr. Pierce, that the radical surgery of these sinuses is very much abused. Acute infections of the sinuses usually recover spontaneously. In operating upon chronic cases, the conformation of the head should be studied and the operation should be done so as to promote free drainage. With proper drainage, the natural tendency of many cases is to recover.

Dr. Wishart: I agree with Dr. Roe that free drainage is a very important factor in the treatment of chronic inflammation of the sinuses.

In regard to atrophic rhinitis, the more cases I see, the more I realize the importance of considering sinus disease as an etiological factor. Each case of atrophic rhinitis should be investigated carefully. One year ago, I saw a case of atrophic rhinitis with no apparent disease of the sinuses. I gave the patient directions in re-

gard to thoroughly cleansing the nose and did not see him again for a year. Then the crusts had all disappeared, but pus could be seen in the neighborhood of the right sphenoidal sinus. This sinus was infected, and establishing free drainage here, practically cleared up the trouble in the nose.

Dr. Phillips: We should differentiate the two types of sinusitis, acute and chronic. Acute sinusitis tends to get well; chronic sinusitis does not tend to recover until free drainage is established. I do not believe that chronic infections of the sinuses ever recover without operative procedures.

Dr. Makuen: We should endeavor to find out the cause of these diseases. As Dr. Kyle said, the interference with the nerves and the blood supply is important. The development of the bone, mouth breathing, adenoids, irregular septa, and the shape of the turbinated bones, all of these things interfere with the development of the frame work of the nose, and of the accessory cavities.

Dr. Coakley: In reference to the paper read by Dr. Beck: Several things are to be considered in studying the radiographs of the accessory sinuses; the thickness of the bone, the size and position of the cavities and the condition of the mucous membrane lining them, all have some influence upon the radiographs and it needs careful training and study to properly interpret the results. My experience in opening the antra of patients who have atrophic rhinitis has shown me that the antra are usually small in size and the walls are very much thickened. The mucous membrane is also thickened. In a number of cases I have found no pus in the sinuses.

Unless the radiographs are clear and distinct, they are not reliable for the purpose of diagnosis. You must have good, clear plates; poor plates may help to confirm your suspicions and upon operating, you will be chagrined to find the cavities normal.

Dr. Beck: The sinuses are frequently involved in atrophic rhinitis, and when treatment is directed to them, better results are obtained. The skiagraphs taken by Dr. Caldwell, which Dr. Coakley has exhibited, are the best that I have ever seen and while mine are not so good, I do not think they are altogether unreliable.

PAPER:

CONCERNING THE RADICAL TREATMENT OF CHRONIC DISEASES OF THE ANTRUM WITH SUGGESTIONS FOR A NEW METHOD IN OPERATING BY THE SUBMUCOUS RESECTION OF THE LATERAL NASAL WALL.

R. BISHOP CANFIELD, M. D.

The chief objection to the generally accepted radical operative procedure for the relief of chronic disease of the maxillary antrum, i. e., the Luc-Caldwell operation, to my mind has been that it offered no opportunity for visual control of the operative field during the after treatment. To review briefly the steps in this operation, it consists in:

1. A mucous membrane incision above the alveolar margin and retraction of the mucous membrane and periosteum.
2. The removal of the anterior antral wall.
3. More or less complete curettage of the lining membrane of the antrum.
4. Formation of a counter opening into the nose, by removal of a part of the lateral wall of the nose.
5. Formation of a mucous membrane flap from a part of the lateral wall of the nose to insure permanence of the opening into the nose.
6. Suturing of this flap in place to the floor of the antrum.
7. Packing the antrum with gauze, the ends of which extend into the nostril.
8. Suturing the initial incision for primary union.

Five to eight days later, the gauze is removed through the nostril and the after treatment is carried out through the nose by means of irrigation, insufflations, etc. By this method many cases pass through a brief convalescence to a satisfactory termination. Many however, fail of a complete cure. Failure has been ascribed to many causes, such as:

1. Great size of the antrum.
2. Essentially chronic character of the disease process.
3. Marked degree or character of the pathological change.
4. The extent of the pathological process which may have involved all or nearly all the other accessory cavities.

In many cases failure has been due, without question, to one or all of these causes. It has seemed to me, however, that if a satisfactory and thorough operation has been performed, and all or nearly all of the disease eradicated, cure should result in cases where failure has been recorded.

Opportunity has been afforded me to reopen antra that have been operated upon radically by the above method, and I have found in some of them irregularities in the healing process which, although sometimes distinctively reparative in their nature, have been instrumental in prolonging convalescence or even making a cure impossible, in some places exuberent and purulent granulations or infected epidermis, in others depressions in a thickened mucous membrane which contain pus. Occasionally there are seen bands of adhesions that more or less completely wall off sections of the antrum and divide it into chambers, one of which may be healing, while others contain pus.

Until recently, the only method by which the antral cavity could be kept under observation after operation was by leaving the initial incision open and carrying out the after treatment through the canine fossa until healing had progressed to such a point that the initial incision might safely be sutured and treatment carried out through the nose. Many objections have been raised to such a procedure:

1. The after treatment is painful.
2. The wound must be kept packed.
3. The patient's saliva is kept saturated with iodoform.
4. Reinfection from the mouth secretions continually takes place.
5. Deformity of the cheek sometimes occurs.
6. Subjective sensations of pain, cold and discomfort frequently persist as long as the opening is allowed to remain.

Friedrich, in 1905, was the first to depart from the accepted method by making a skin incision around the ala. Through this incision he removed the anterior antral wall, the lateral wall of the nose and the lateral wall of the pyriform opening together with the anterior half of the inferior turbinate. He then packed the antrum

with gauze and allowed the ends to extend into the nostril in the usual way. The skin incision was then sutured for primary union. Of course objections to this method of operating are readily seen. It offers danger of scar and of wound infection and increases the gravity of the operation.

Kretschman made the second advance by doing the ordinary Luc-Caldwell operation through the usual alveolar incision, and in addition removing the lateral wall of the nose as far as the lateral wall of the pyriform opening. This he left standing for fear of deformity. By this method he hoped to secure some view of the antrum throughout the convalscence, but as one can readily demonstrate by doing the operation, he failed.

Later Denker made a decided advance by doing the Luc-Caldwell and in addition removing the lateral wall of the nose, including the lateral wall of the pyriform opening. The flap was cut in the usual way and the antrum packed. The initial incision was sutured. This operation converts the nose and antrum roughly into one cavity, of which a fairly comprehensive view can be secured through the nose. He reports three cases, in the first of which he was not successful on account of the presence of a complication involving the frontal sinus. The patient was, however, much improved. In the other two cases, the patients were very greatly benefited, the only remains of the trouble being a trace of moisture upon washing.

I have operated seven cases somewhat after this method. In the first three, I followed it as closely as might be. In the last four, I varied it as follows:

1. Incision through the alveolar mucous membrane to the median line.

2. Retraction of the soft parts.

3. Removal of the lateral wall of the pyriform opening and the lateral wall of the nose backwards for about two-thirds of the distance of the attachment of the inferior turbinate, and upwards from the floor of the nose to and including the attachment of the inferior turbinate.

4. Removal of as much of the anterior wall of the antrum as seems necessary.

5. Formation of the mucous membrane flap which is turned down into antrum.

6. Packing the antrum with gauze, the ends of which extend into nostril.

7. Suturing of initial incision for primary union.

Such an operation should be preceeded about two weeks by a preliminary amputation of the anterior half of the inferior turbinate. The technique offers a few difficulties not encountered in the Luc-Caldwell, chief of which is the thorough removal of the nasal wall along the attachment of the inferior turbinate. It presents several advantages:

1. It offers a satisfactory view of the antrum through the nose during the after treatment.

2. It permits careful observation and control of the healing process.

3. It can be made as radical as necessary by removing the entire anterior wall and entering the ethmoid and sphenoid, or it can be made a comparatively conservative operation by removing less of the anterior wall of the antrum and the lateral wall of the nose if, at the time of operation, closer examination of the condition renders the more extensive operation unnecessary.

4. The after treatment is painless.

5. The greater part of the after treatment can easily be carried out by the patient.

I wish briefly to report the following seven cases:

Case 1. Mrs. D., 40 years. Came to hospital on account of antrum disease which began five years ago during an attack of grip, with pain and swelling of the side of the face. Pain was much worse in the morning. During the afternoon profuse discharge would appear in the nostril. Pain lasted for about five weeks, but discharge persisted. At this time, a healthy first molar was extracted in order to establish communication between the mouth and the antrum. A slender gold tube was inserted and held in place by means of a silk thread fastened to an adjoining tooth. This tube she has worn ever since. She has had occasional exacerbations of the trouble. Five days ago she lost the tube in the antrum. Transillumination positive.

Examination. Nose (right) strings of pus throughout the nose and the post nasal space. Polypoid degeneration of the anterior end of the middle turbinate. Mouth in fairly good condition. Culture from antrum shows streptococcus. X-Ray discovers the foreign body in antrum.

March 14, 1906. R. Radical antrum operation; ether 1½ hours. Antrum of great size filled with stinking pus. Mucous membrane shows marked polypoid degeneration. Curettage of entire cavity. Removal of greater part of the anterior antral wall and of the lateral wall of the nose, including that of the pyriform opening in its

lower half. Foreign body recovered. Sinus through tooth socket closed.

March 18. Gauze and stitches removed. Slight amount of pus.

March 31. Patient discharged. Antrum dry. Given tube for necessary irrigation.

May 31. Returned for control. Antrum dry. Practically no discharge since leaving hospital.

Case 2. D———, laborer, 29 years, Kalamazoo, Mich. Comes to hospital on account of discharge of pus through socket of an extracted tooth. This discharge has persisted since the tooth was removed a couple of years ago.

Examination. Moderate hypertrophy of inferior turbinate. Some oedema of middle turbinate. Pus in middle meatus and generally throughout the nose. Sinus leads upwards through the socket of the left first molar tooth. Pus appears through this sinus.

March 2. Anterior half of left inferior turbinate removed.

March 16. Culture from antrum shows streptococcus.

March 16. Radical antrum operation, left. Ether, 1½ hours. The radical operation was performed with removal of the lower half of the bony wall of the pyriform opening. Antrum enormous and contains fair amount of muco pus. Lining membrane shows marked hypertrophy with some polypoid degeneration. It was thoroughly curetted. Usual mucous membrane flap, antrum packed with gauze and initial incision sutured. Sinus through tooth cavity curetted.

March 21. Gauze removed. Slight amount of pus.

April 10. Patient given a Halle canula and allowed to carry out daily irrigation. Occasional observation of antrum through nose with applications to healing surface.

May 11. Finger passed through nose into antrum shows everywhere lining membrane perfectly smooth. Antrum is apparently perfectly healed, although as every effort to close the sinus through the tooth cavity has been unsuccessful there still remains some secretion which reaches antrum from mouth.

Case 3. Mrs. M. D———, 36 years. Two years ago during an attack of grip contracted an attack of acute disease of the left antrum. Condition was allowed to become chronic. A year ago first upper molar was extracted, communication established between antrum and nose and a gold tube inserted. This tube she has worn ever since. She has continual discharge with some pain and swelling of cheek and eyelid despite daily irrigation.

Examination. Moderate oedema of anterior end of middle turbinate. Small amount of pus on posterior end of inferior turbinate. Probe passed through sinus in tooth cavity reveals thickened mucous membrane, with granulations high up and far back. Transillumination positive. X-ray shows slight diffuse haze over entire antral region.

May 13, 1906. Radical antrum operation. Ether, 1¾ hours. Mucous membrane of antrum shows marked polypoid degeneration about upper end of sinus. Far back beneath orbit were several polyps and some pus. Antrum and ethmoid thoroughly curetted. Lateral wall of nose together with part of bony wall of the pyriform opening removed in the usual way. Usual flap, packing and suture of initial incision. Sinus through tooth curetted.

May 18. Gauze removed.

June 10. Under daily observation through nose, antrum has healed. For several days has been no pus.

June 23. Remains dry.

Case 4. A———, laborer, 31 years, Raber, Mich. Comes to hospital on account of discharge from right nostril which has persisted for four years without other symptoms. Four years ago contracted syphilis, which has been throughly treated without appreciable effect on the antral condition.

Examination. Considerable oedema of entire nasal mucous membrane. Marked oedema of both inferior and middle turbinates. Right side shows middle meatus filled with crusts and pus. Nasopharynx also contains considerable amount of pus and dry secretion. Transillumination positive. X-Ray indefinite.

April 24, 1906. Radical antrum operation, right. Ether. 1½ hours. Operation performed in usual way with resection of anterior antral wall, lateral nasal wall and bony wall of pyriform opening. Flap formation, packing and suture. Antrum was found to contain small amount of pus having a somewhat foul odor. Median wall of antrum was dark, soft and rather sugary, evidently carious. Thorough curettage of antral lining and of ethmoid.

May 2. Gauze removed.

May 10. Developed metastatic thrombo-phlebitis.

May 30. Antrum dry. Considerable secretion in nose in the neighborhood of the ethmoid. Patient disappeared.

June 15. Antrum dry. Small amount of secretion in ethmoid. Patient disappeared.

July 15. Patient reappeared. Antral mucous membrane smooth and cavity apparently dry. Some flakes of secretion appear on washing.

Case 5. Miss H. Nurse. Has had discharge from left nostril with occasional pain in left cheek since attack of grippe about two years ago. At times there is some odor in nostril.

Examination. Hypertrophy of both inferior turbinates. Fair amount of pus in left nostril and in nasopharynx.

Transillumination positive. X-Ray questionable.

August 9, 1906. Left radical antrum operation. Ether, 1¼ hours. Operation in the usual way with resection of anterior antral wall, lateral wall of nose and lateral wall of pyriform opening, pack-

ing, flap and suture. Antrum contained much creamy pus, mucous membrane showed considerable thickening.

August 17. Gauze removed.

August 30. Patient has had daily irrigation with occasional application of silver nitrate solution, 10 gr. to the ounce. Slight discharge.

October 1. Antrum has been dry for some time.

Case 6. Mrs. F. Trouble began four years ago with headache, soreness, sense of tension in head and in right cheek. No swelling or discharge until opening was made through socket of first molar tooth which tooth was extracted for the purpose. Since then has had continual discharge through tooth cavity with frequent swelling and pain in cheek and lower eye lid. Two years ago had a nose operation on the right side.

Examination. Right. Anterior end of middle turbinate gone. No pus in nose. Marked hypertrophy of inferior turbinate. Sinus leads up through socket of first molar tooth. Irrigation through sinus allows pus to escape through nose. Patient complains of considerable pain in region of antrum and above right eye, which persists almost without intermission. Transillumination positive for antrum and frontal sinus.

December 4, 1906. Radical antrum operation. Ether 1½ hours. Operation in the usual way with resection of anterior antral wall, lateral wall of nose and pyriform opening. The antrum was found filled with pus and thickened membrane to such an extent that cavity was almost obliterated. Cavity thoroughly curetted, ethmoid entered, found filled with infiltrated membrane and obliterated. Usual flap, packing and suture. Tooth sinus curetted.

December 7. Gauze removed, free escape of pus. Satisfactory view of antrum secured through nose.

December 20. Daily treatment, pus in slight amount from antrum, but in considerable amount from frontal sinus.

January 15. Antrum apparently almost healed. Still considerable pus from frontal with pain and headache. Killian operation advised.

February 10. Apparently very slight amount of pus in antrum which seems to be lined with a smooth membrane. Still pus from frontal.

June 20. Antrum healed. Has been dry two months. Some pus from frontal.

Case 7. Mr. J., dental student, 28 years. Has had discharge from antrum for about three years. In the beginning, it was supposed to come from a diseased first molar, so the tooth was removed and communication established through its socket between antrum

2. Infiltration anasthesia with ¼% cocain in 1-5000 adrenalin and mouth. Since then continual discharge from mouth and from nose.

Examination. Nose negative except for small amount of pus on nasal floor. Sinus through second molar through which pus appears. Probe passes up into a small antrum and discovers soft granulations. Two weeks treatment was quite without result.

January 14, 1907. Radical antrum operation, left. Ether, 1 hour. Usual operation with resection of lateral wall of pyriform opening, lateral wall of nose and part of anterior wall of antrum. Antrum found to be very narrow, but fairly deep. It contained pus and degenerated lining membrane considerably thickened and degenerated. Ethmoid was entered, found involved and curetted. Usual flap, packing and suture.

January 18. Gauze removed.

Febuary 18. Very slight amount of secretion in antrum when washed. Ethmoid area shows some crusts and secretion.

May 1. Dry.

In looking over this short series of cases I feel that the results are more satisfactory than those secured by the Luc-Caldwell operation. After the latter operation the subsequent treatment must be carried on largely in the dark, and the condition of the antrum inferred from the amount of discharge and the severity of the symptoms. After the operation just described treatment can be carried out under visual control, the healing process watched with ease and the final result looked forward to with some degree of certainty.

The above operation then offers a fairly satisfactory way of dealing with chronic antral disease, either simple or combined. It has seemed to me, however, that an operation that would afford free access to all the accessory sinuses without an incision through the buccal mucosa would offer many advantages. My last case, therefore, I have operated upon entirely through the nose by a submucous resection of the lateral wall of the nose, as follows:

1. Preliminary amputation of the anterior half of the inferior turbinate two weeks, if possible, before the antrum operation.

2. Infiltration anesthesia with ¼% cocain in 1-5000 adrenalin injected along the lateral wall of the nose and the anterior wall of the antrum.

3. An incision through the soft parts of the nostril at the junction of the skin and mucous membrane, beginning about the middle of the lateral wall of the pyriform opening and carried well down to the floor of the nose. This incision passes through the periosteum to the bone and corresponds exactly to the lateral wall of the pyriform opening.

4. Elevation of the mucous membrane and periosteum of the lateral wall of the nose from the floor of the nose to the level of the insertion of the inferior turbinate and backwards as far as the inferior turbinate has been amputated.

5. Elevation of the periosteum of the anterior antral wall for any desired distance.

6. Removal of the inferior half of the lateral wall of the pyriform opening and the lateral wall of the nose over an area corresponding to that part from which the mucous membrane has been elevated.

7. Removal of the desired amount of the anterior antral wall, i. e., enough to allow view of antrum.

8. Penetration of the lining membrane of the antrum and inspection of the cavity.

9. As thorough curettage of the antrum as necessary.

10. Curettage of the ethmoid and entrance of the sphenoid if indicated.

11. Formation of a flap of the mucous membrane of the lateral wall of the nose with base downwards.

12. Application of this flap to the floor of the antrum and packing with gauze.

There seems to me to be several advantages in this operation.

1. It can be performed under local anaesthesia.

2. It offers a most satisfactory view of the antrum and maintains it throughout the after treatment.

3. It can be made just as radical or just as conservative as may seem wise as the operation proceeds.

4. It offers the most direct and adequate approach to the ethmoid and sphenoid.

5. It necessitates no buccal incision.

6. It requires no suture.

7. It simplifies the after treatment greatly.

8. It accomplishes all that any other operation can in similar condition.

The difficulties of the operation are mainly those encountered in the sub-mucous resection of the nasal septum, increased somewhat by the fact that the lateral wall of the nose is sharply concave and that the bone of the lateral wall of the pyriform opening is very dense and requires the energetic use of the mallet and chisel or a strong rongeur.

I wish to report the following case:

Mr. S., 23 years, Jackson, Mich. Came to hospital on account of a discharge from the left nostril, which has persisted for some years. Early in the course of the disease the first molar was extracted and communication was established between the antrum and mouth. Through this opening the antrum has been irrigated daily every since without improvement.

Examination. Moderate spur. Left nostril contains pus. Considerable hypertrophy of both inferior and middle turbinate. Probe passes through sinus in the socket of the previously extracted tooth into antrum. Mucous membrane of antrum as felt by probe seems to be soft and velvety. Pus follows the probe.

Operation by the above method allowed free view of antrum, which contained much pus, granulation tissue with some polypoid degeneration. Ethmoids were easily accessible, were found filled with thickened mucous membrane and some pus and were curetted. The flap formed of the lateral wall of the nose was torn so that about half of it was lost. The remainder was turned down over the floor of the antrum, which was then packed with gauze.

I was much pleased with the operation on account of the ease with which the antrum was opened and examined. Almost the entire antrum could be satisfactorily seen. Even far back under the orbit, a region always somewhat hard to see, was clearly visible. With the anterior half of the inferior turbinate and corresponding part of the lateral wall of the nose out of the way the ethmoid was thoroughly accessible and was curetted with more satisfaction than I had ever experienced before when attacking it from below.

(Since reading this paper other operations have been *performed* according to the last mentioned method with satisfactory results.)

DISCUSSION.

Dr. Coakley said: The paper presented by Dr. Canfield is very interesting. It seems to me a most excellent operation. In the majority of cases, a general anaesthetic would be preferable to local anaesthesia, because it would be impossible to do a complete operation without causing severe pain.

PAPER:

THE ACOUSTICS OF THE MOUTH AND THE RELATION OF THE INDIVIDUAL'S VOICE TO HEARING.

D. BRADEN KYLE, A. M., M. D.

Much has been written from a physiologic standpoint concerning the speaking and singing voice. Such writers and investigators as Mueller, Helmholtz, Gradenigo, Hensen, Gruber, Bezold, Spear, Bonnier, Rossback, Merkel, Cohen, Makuen and Mackenzie have added much to our knowledge of the mechanism of phonation and hearing. Yet, after all, while a thorough knowledge of the normal condition is essential, the various *methods* used in training the voice are necessitated by pathologic alteration involving some portion of the speaking tract.

The *normal* condition would only require *training* in executing, while the *pathologic condition* causes *straining* in execution. The anatomy and physiology is, of course, important, yet if all the tissues and parts were normally formed there would be little difficulty in training the human voice, but in the majority of cases there is some deviation from the normal which interferes with the regular mechanism of the apparatus of phonation. It is with these irregularities or pathologic conditions that we purpose to deal. It is of the greatest importance that the teachers of elocution and music should thoroughly understand these irregularities and the very fact that *different* teachers *strongly* and *urgently* uphold different methods proves the existence of such irregularities. If the formation of the vocal apparatus was always normal there would be no necessity for methods. This explains why some methods are successful in some individuals and failures in others.

The method, then, should be adapted to the individual case and not the individual case to the method. The use of the facial muscles in a variation of tone is merely the power to expand the walls of the building, increase the volume and lessen the resistance of tone. The power to depress the tongue and throw it in any position

desired voluntarily lowers the floor of the building, increases the space of outlet and enables the individual to have a larger compass of voice.

Voice production requires the use of a complicated mechanism, i. e., the so-called musical ear. Through conscious or unconscious cerebration there are called into action for voice production three anatomical factors—the lungs, the larynx and the resonance tube. The resonance tube includes all structure above the vocal cords, which includes the vestibule of the larynx. The pharynx, the tonsils, the posterior nares, the anterior nasal cavities, the accessory sinuses, especially the antrum of Highmore, and the mouth. It is this resonance tube that after tone is produced modifies or aids the fullness, the smoothness, the roundness, the power, the sweetness and beauty of the voice. It is in this tube that the sounds produced in the larynx are re-enforced and it is in this same resonating tube that these sounds may be distorted and converted into rasping, disagreeable tones.

Now, if the three essential anatomical factors, namely, the lungs, larynx and resonating tube, are proportionate then the tones coming from the larynx, being modified by the perfect resonator, unite and modify each other. If, however, there is any disproportion this resonator acts as a distorter of sounds. It is like a tenor string on a bass violin, it is a misfit. If all parts work harmoniously that individual is gifted with a natural voice and is a natural singer, requiring only careful education and practice to make perfect execution, but when such harmony does not exist, where diseased conditions or imperfectly developed parts in the voice apparatus do exist, it is then that the teacher of music must differentiate the condition, as one will require a *method* and the other merely *execution.*

Volume, tone and timbre are controlled by the size of the lungs, the larynx and the resonance tube. Their loss then may result from (1) disease of the lungs, bronchia or trachea; (2) the larynx, with its innervation, not only local lesions, but lesions elsewhere; (3) diseases of the pharynx and tonsils; (4) diseases of the nose, including septum and antrum.

Each voice has its individuality, in fact, our voice is a part of our individuality. Tone production, timbre or quality and resonance together with execution do not repeat themselves in the

same manner in two individuals. This is especially marked in singers.

As to the question then of the acoustics of the mouth in its relation to the voice, the upper part of the resonating tube—the mouth, the tongue, the hard and soft palate, naso-pharynx, nostrils and accessory cavities bear the same relation to the voice as the building does to the speaker. The voice may be produced correctly, but its quality lost in faulty acoustics.

The building may be beautiful architecturally, but its acoustic properties poor. The decorator may improve the acoustics of the building, so may the laryngologist and rhinologist improve the acoustics of the mouth by correcting faulty conditions that interfere with perfect resonance. For example, the removal of enlarged tonsils, naso-pharyngeal growths, correction of nasal obstruction, etc. Again, in some cases, certain pathological conditions may improve the acoustics. One of our famous singers has abnormally large tonsils, in fact so large that when the tongue is protruded and the muscles of the larynx made tense, the tonsils project so as to meet in the median line; yet, when that individual sings, by depressing the tongue the tonsils fill into the lateral cavity of the pharynx, the patient having an unusually wide pharynx with concave walls. In this position, then, the tonsils offer no obstruction to sound in the resonating tube, while the removal of these tonsils would entirely alter the walls of the building and would also alter its acoustic properties.

An advantage of the mouth as a part of the resonating tube and its relation to acoustics is that the soft parts, namely, the cheeks and the tongue can be placed in various positions at will, so that the walls of the building can be expanded or contracted and the floor of the building elevated or depressed at the will of the individual.

The relation of the voice to hearing and the control of the voice by hearing presents an interesting subject for discussion and study. *Voice* is not *hearing;* we also have voice in the absence of hearing, yet *hearing* is really the *controlling* element in the production of voice. The relation of our own hearing to our individual voices is illustrated in our speaking and in singing. Our voice to us is what our ears indicate.

As to hearing, this necessitates two classifications, namely, *subjective* and *objective* hearing. By subjective hearing we mean the

individual's subjective sense of sound-perception; in various lesions of the ear with the various noises heard by the patient, that is subjective; his sense of sound-perception of his own voice is subjective. However, in noises of the ear, which are usually only heard by the patient, we can form no comparison, because the observer cannot hear the noise. As to his own voice, we can compare his subjective sense of sound-perception with that of the observer. The individual's subjective sense of sound-perception is determined purely by external sounds. As to whether this is normal or abnormal he can easily determine by comparison with the external objective sense of sound-perception of others.

The training and cultivating of the voice under the guidance of a teacher, to be sure, is a great factor in the success of the individual as to his speaking or singing voice; however, one of the greatest difficulties the instructor often has is to convince the pupil that his voice is wrong. The pupil's own ears tell him that it is right. Many of you probably are aware that one of our greatest singers has one note which to that individual's ears is in perfect harmony, but which in reality is one-half tone flat.

A musical ear does not always mean a musical voice. Some individuals cannot sing or play and, as far as the music is concerned, could not tell "Yankee Doodle" from "Old Hundred;" yet the slightest discord in the human or orchestral tones will be detected instantly by such a person's hearing.

Again, certain individuals may have a musical voice as far as conversation or speaking is concerned, I say musical, meaning a pleasing tone, yet such individuals cannot sing and their ear will not tell them whether they are in chord or dischord. If, however, they are attempting to sing with others their ear will tell them if the voices do not harmonize, yet they cannot control it, as their ear does not seem to indicate to them that fineness or distinction of tone which permits of harmony.

The alteration in the individual's voice where the hearing has become defective is so marked as to become almost characteristic, although there are exceptions to this. I have seen a number of cases in which the patient was so deaf that he could not hear conversation even in the very loudest tones, yet there was practically no alteration in the voice, but this is, indeed, the exception.

The deaf and dumb may be taught to speak, yet the voice produced is unnatural and not altogether pleasing. The hearing, then,

associated with the voice acts as a regulator; defective hearing may mean altered voice.

It often falls to the lot of the laryngologist to examine the throat of singers or would-be singers, and many times the instructor of music, having used all his methods and means to train a certain voice, finally appeals to you to determine what is the matter. The pupil is thoroughly convinced that he can sing, and to his ear he can sing, but, unfortunately to the nine hundred and ninety-nine listeners, his subjective sense of sound-perception is faulty. These cases are, indeed, pitiful. I do not mean that we do not have some cases who think they can sing in spite of their hearing and voice. These facts must be taken into consideration by the elocutionist and the teacher of music.

Another extremely interesting fact illustrating the relation of the voice and hearing is this; in the first place, few of us could accurately describe our voices so that the voice would be recognized by any one else. Frequently we hear individuals discuss voices, either speaking or singing, and while seven out of ten might agree that the voice was pleasing, melodious, soft and sympathetic, and possessed the many other attributes necessary in a successful singer, yet the remaining three of the ten would find fault, some rasping note, something not pleasing, showing that the objective sense of sound-perception varies greatly in individuals. Frequently individuals are criticised for their loud tone of voice. Individually they may be charming, but their loud tone of voice frequently attracts attention. Did it ever occur to you that this indvidual himself did not know that he was speaking in such a loud tone of voice? I know this is true, as I have interviewed several such persons, who had been taken to task for their loud tone of voice. When I would have them lower their voice to an ordinary pleasing conversational tone, they assured me that to them it sounded as though they were speaking scarcely above a whisper, showing that their ears for their own voice were not so sensitive as for outside sounds. In such individuals the subjective sense of sound-perception was decreased or below normal, while their objective sense was normal. On the other hand, some individuals who use a quiet, soft, low tone of voice, to their own ear it sounds as if they were speaking in a very loud tone of voice. In such individuals their subjective sense of sound-perception is extremely sensitive or exaggerated. Just as objective sense to sound may vary, so does the subjective sense vary.

The effect of drugs and stimulants also illustrates the peculiar relation of the individual's voice to hearing, in that certain drugs or stimulants may exaggerate the two conditions which I have just described. The man with the defective loud voice talks louder and the man with the defective low voice finally speaks so low that you can scarcely hear him.

The jerky, irregular voice of deaf person is another index or evidence of the peculiar relation of the voice and hearing. Without hearing, of course, there is no sound, but the voice is more than sound, and the voice to the individual or rather the individual's voice to himself, is exactly what his ear tells him it is, and if there is any loss of harmony between these two, then he will have defective voice, yet in response to all the tests of hearing he shows normal reaction.

A few days ago, in training a young man, whose voice had not changed at puberty, and finally by the use of the falsetto voice I had worked him down to a perfectly natural tone and after having him speak in that one for a few minutes his face assumed an anxious expression and he said, "Will that be my natural voice?" for to me it sounds frightfully strange and unnatural. His ear had not been trained to that sound.

Pathologic alterations of the structures of the naso-pharynx, whether due to local or systemic changes, will produce subjective and objective alterations in the sound-perceiving apparatus. The objective one can be determined by tests, while the subjective ones can only be described by the individual. The tinnitus associated with any such alteration, no matter what form it may assume, is heard only by the individual, except in rare pulsating cases, and it cannot be detected by the observer, although frequently subjective sounds are so intense that the individual thus afflicted can scarcely realize that the sounds cannot be heard by the observer.

The involvement of the apparatus of subjective sound-perception, especially of the inner portion or the orifice of the Eustachian tube, will give to the patient the sensation of altered voice, common in singers. To the audience their voice is in perfect form, while to their own ear the voice sounds muffled, in other words, the subjective perception is interfered with, while the voice is in perfect form. At the same time, this indvidual may not show any defective objective sound-perception. In objective sound-perception the external ear is the collector of sound, while the drum membrane and ossicles of the middle ear are the transmitters of sound; in sub-

jective hearing, this condition is partially reversed, the Eustachian tube partially taking the place of the external ear.

Sound is what we hear; our perception of sound depends on whether it is subjective or objective and our description of sound will depend on the condition, subjective and objective, of our sound-perceiving apparatus. The deaf-mute has no conception of perception of either subjective or objective sounds.

Voice is sound, speech is voice in action; the impression made by voice and speech will depend entirely upon the condition of the sound-perceiving apparatus; to the individual himself it will be both subjective and objective, but to the listener it will be entirely objective. The impression given to either speaker or hearer, will depend upon the acoustics of the mouth and the condition of the subjective and objective hearing of the individual.

DISCUSSION.

Dr. Pierce: In regard to the importance of the nose and nasopharynx and pharynx as resonator cavities, I think it is very difficult to ascribe to them their proper influence in the production of the voice, from a simple examination of the throat. Not long ago, one of the leading tenors of the world came to me early in the morning saying that he wanted to sing in "Faust" that night. I examined his throat and found his tonsils very much enlarged; the vocal cords acutely congested and the nose also inflamed. I advised against his attempting to sing that night but he waived aside all my suggestions and so I did what I could under the circumstances, I heard him sing that night; his voice was beautiful with good staying powers and clear tones. This simply illustrates what some people can accomplish in the use of their voice in spite of adverse conditions.

In some singers, the removal of the tonsils will improve the voice; in a few cases it has been known to change the tone of the voice. Other things being equal, it is better to have a thoroughly healthy nose, throat and larynx but after a good singer has trained the voice and secured good results in spite of some pathological conditions, it is probably better to avoid radical measures in dealing with these conditions.

Dr. Stucky: I should like to ask Dr. Kyle if he knows of any instances in which the voice has been lowered by the removal of the tonsils. In my experience of twenty-five years, I have known of

three cases; one man's tenor voice was lowered one-half a tone. Two women each claim that the tone of their voices has been lowered. McKenzie mentioned this as a possible danger.

DR. ROE: I have had a few cases in which the tones were made higher by operating upon the tonsils.

DR. BALLENGER: Many cases have been reported in which the removal of the tonsils has improved the quality of the voice. This is undoubtedly so in the majority of cases. In a few cases the tone is probably lowered slightly.

PAPER:

TWO CASES OF EXTENSIVE CHOLESTERIN INFILTRATION OF THE MASTOID CELLS.

EDWARD BRADFORD DENCH, M. D.

Cholesteatomatous deposits within the middle ear, as the result of chronic suppurative inflammation, are by no means uncommon. In almost every case of chronic suppuration, in which a radical operation is performed, cholesteatomatous masses, varying widely in size, are found in the tympanic vault. Exceptionally, these cholesteatomatous masses involve not only the tympanic vault, but to a greater or less extent, the entire mastoid apophysis. Under these conditions, the cholesteatoma consists of a mass of superimposed epithelial cells forming a tumor which, by its gradual increase in size, has caused the absorption of the bony trabeculae separating the mastoid cells, until the entire mastoid process is converted into one large cavity. In this epithelial mass we find a certain amount of cholesterin. In the cases which I am about to report, however, the true cholesteatomatous tumor was insignificant in size But in each instance, all of the mastoid cells were infiltrated with cholesterin.

The first case was that of a young woman, 17 years of age, who had had a suppurative otitis upon the right side for twelve years. At no time had the suppuration been very profuse. The patient had

tented himself with keeping the ear thoroughly cleansed, and as the patient had had no acute symptoms, operative interference did not seem imperative. As the discharge continued, however, at intervals, he finally referred the case to me.

The patient gave a history of having had occasional attacks of vertigo and recurrent attacks of discharge from the right ear at intervals. There was a history of some mastoid tenderness. When I saw the patient, the temperature was normal, there was no pain in the ear, although pressure over the mastoid elicited considerable tenderness. The patient was somewhat nervous, and it should be stated that pressure over the opposite mastoid was also painful.

In view of the history in this case, a radical operation was performed, and a small cholesteatomatous mass was found in the mastoid antrum. The mastoid cells throughout were deeply congested. The cells below the antrum, down to the very tip, presented a peculiar appearance. Instead of the glistening lining characteristic of the healthy mastoid cells, the lining membrane of these cells whereever exposed, was dull in color, and the bone when removed either with the gouge or rongeur, did not present the characteristics of healthy bone. I can compare the texture of this bone to nothing but that which might be found if the normal mastoid cells were filled with wax. In the course of the operation, it was necessary to ablate the entire mastoid process.

From the appearance of the bone, I was inclined to believe that the process was a tubercular one, and consequently sent several specimens to the laboratory for examination. The pathologist's report shows an infiltration of all the mastoid cells with cholesterin, even the cells at the tip being involved. The pathological findings throughout were peculiar, in that cholesterin was found everywhere, but very few germs were found in any part of the mastoid. The condition was one entirely new to me. I have had no opportunity to search the literature upon this subject, but do not remember reading of a similar case.

It seems to me quite possible that a cholesterin infiltration of this character might explain the rapid breaking down of the mastoid process in many cases of chronic suppurative otitis media, which have remained quiescent for a long period, and it is for this reason that the history is reported so fully.

In this instance, the patient made a rapid and uninterrupted recovery after the radical operation.

In the second case, occurring in a young woman of 26 years of age, practically the same condition was found. In this case the otitis media came on after typhoid fever, and had existed for three years prior to operation. There was a history of recurrent attacks of acute suppuration during this period. None of these attacks, however, had been severe. At the time of operation, which was performed on account of the history of recurrent suppurative attacks,—the same condition as detailed in the preceding case, was found, namely, cholesterin infiltration of all of the mastoid cells, with a not very extensive cholesteatomatous mass in the mastoid antrum.

This case also made an uninterrupted recovery after the radical operation.

These cases are reported in detail because, so far as I know, attention has not been called to this condition in otological literature. In operating upon cases of this character, the question naturally arises whether it is necessary to ablate the entire pneumatic structure of the mastoid, where this condition exists. It has ordinarily been my practice, in performing the radical operation for chronic suppurations, to limit the operation to that part of the mastoid which showed gross evidences of disease. While in the majority of cases where the radical operation was performed, the lower mastoid cells were either usually absent or were very small, it has never been my practice where these cells have appeared healthy, to ablate the entire mastoid. As soon as normal cell structure was reached, I have concluded my operation, and the results have always been satisfactory. In the two cases under consideration, however, the cholesterin infiltration of these cells seemed to constitute a peculiar factor, sufficient to warrant the complete ablation of the mastoid. It is possible that these cases might have recovered if the antrum and adjacent cells alone, had been cleared out and the radical operation performed in the usual manner, without removal of the mastoid tip, even although this part of the mastoid contained cholesterin. Personally, I should be unwilling to terminate the operation at this point, and I believe that the condition described may constitute a factor in cases where suppuration persists after an apparently complete radical operation.

All of us who have performed many radical operations, know that this procedure is not uniformly succesful in causing a complete cessation of suppuration. In the majority of cases where slight

suppuration persists after the operation, that portion of the middle ear about the tympanic orifice of the Eustachian tube is at fault. While this condition is annoying, I do not consider it dangerous. There are certain other cases, however, in which although no diseased bone can be detected in the cavity left by the radical operation, slight suppuration continues. It is a question in my mind whether, in some of these instances, certain cells containing cholesterin deposits have not been overlooked.

I believe it wise, therefore, in every instance where the slightest doubt is present in the mind of the operator, as to the fact of the lower portion of the mastoid being normal, to give the patient the benefit of the doubt, and to ablate completely the entire mastoid process so as to eliminate every possible focus of disease. In the cases detailed above, although the operation was very extensive, recovery was exceedingly rapid. One case was practically dry at the end of three weeks, this case being one where primary grafting was employed, and the other case was well at the end of about five or six weeks. In this latter case, owing to the necessary extensive exposure of the lateral sinus, primary grafting was out of the question, and at a secondary grafting, performed on the fourth day after the operation, it seemed advisable to introduce only a meatal graft just sufficient to cover the margins of the enlarged meatus. In spite of these two extensive operations, however, recovery was exceedingly rapid.

From an increasing experience, it seems to me that the more radical we are in these radical operations, the quicker the patients recover.

DISCUSSION.

Dr. Roe: An interesting case of this kind came under my observation recently. There was scarcely any elevation of temperature, slight delirium and a little discharge from one ear. No history of chronic ear trouble; some tenderness over the mastoid. I was in doubt as to any trouble in the mastoid but as there was nothing else to account for the symptoms, I opened the mastoid. There was one large cavity filled with cholesterin. This cavity had a thin wall. I removed the outer shell of the mastoid, cleaned out the cavity thoroughly and then packed it. The mental condition improved immediately and the recovery was uninterrupted.

DR. BECK: I know of several cases of this kind reported in the literature.

DR. BARNHILL: I have operated several similar cases. In such cases I usually remove the tip of the mastoid. I have seen two case of cholesteatoma in which there was an ivory hardness of the mastoid process.

DR. DENCH, in closing: In my paper I tried to bring out the fact that pure cholesterin had been deposited in the cells of the mastoid bone and that the septa between the cells were not broken down, a condition which I have seen only two or three times. Cholesteatoma we see quite frequently. I am familiar with the literature to which Dr. Beck mentions, but it refers to a different class of cases.

PAPER:

ATROPHIC RHINITIS. ETIOLOGY AND TREATMENT. ILLUSTRATED BY RADIOGRAPHS.

JOSEPH C. BECK, M. D.

It is the article of Richards, read at the last meeting of the American Medical Association, that prompts me to bring this subject for your discussion for two reasons: First, he appeals that more attention be given in our medical society meetings to this most discouraging chapter in rhinology; and, second, his ignoring in a measure the sinuses in relation to atrophic rhinitis. I will say at the outset that, believing that the sinuses and their involvement, whether primary or secondary, have much to do with the disease of atrophic rhinitis, I therefore do not believe it to be the most discouraging chapter in rhinology. It is my purpose to report a number of cases of this affection in which radiographs were taken to determine the involvement of the various accessory sinuses of the nose. And by operation that fact was proven.

I also wish to report in connection with these cases, which are 24 in number, several methods of treatment which have not been

brought out very extensively in the literature, some, so far as I am able to find, original and of benefit in this affection.

The literature on atrophic rhinitis is so vast, and you are all so well acquainted with it, that I will not take it up, only in so far as it relates to cause and treatment, and that very briefly.

Zaufal, Heymann and Hopmann are of the opinion, based on many investigations, that the cause lies in a congenital atrophy of all the bones of the nose, and a lack of vitality of the mucous membrane. These individuals are, as a rule, predisposed to catarrhal conditions.

E. Fraenkel, Lowenberg, Abel, and Hajek have found the cocoa bacillus and the bacillus mucosus as the probable cause of this affection. Schlafrig claims that the sarcina, and Belfanti and della Vedova, the pseudo-diphtheria bacillus. One of the most interesting observations is that of Perez, carried on at the Pasteur Institute, where he claimed to have found the pathogenic micro-organism called the cocoa bacillus fœtidus ozenæ. He injected pure cultures of these micro-organisms into the veins of a rabbit, and produced marked atrophic changes in the anterior turbinated body, which corresponds to our inferior turbinated body, much the same, when examined histologically, as in man.

Most of the authors still believe that the primary cause is some violent infection of the nose, such as accompanies scarlet fever, measles, diphtheria, which goes on into a chronic hypertrophic condition, and, due to some associated general malnutrition, with eventually the development of the specific micro-organisms on such good soil, the atrophy develops, and goes on rapidly or slowly, according to the resistance of the tissues and medical interference.

Zerniko believes that this affection is due to a trophoneurotic change in the mucous membrane and bone.

Stoerk believes that congenital lues or gonorrhea in the parents is responsible for many causes.

Recently much attention has been given to the involvement of the sinuses and their chronic discharge as being responsible for this affection.

Michel, Hajek, Gruenwald and many others are of this opinion.

Post-mortem examinations made by Wertheim, Oppinkofer, Krause, Hartmann, Harke and Fraenkel, would all disprove that the sinuses have anything to do with the causation of atrophic rhinitis. Gruenwald, Hajek, and others, however, answer them by saying

that, as a rule, anatomists do not make a very careful examination of these cells, and that in many cases the mucous membrane does not show great changes macroscopically. Again, it is not necessary that all or many cells need to be affected. One or two of the ethmoidal cells only on each side can cause an atrophic condition. From my reading of Hajek's 12 cases, and his deductions, I am convinced that atrophic conditions can be caused by affections of the sinuses. Besides, he has reported two cases of marked hypertrophy of the adenoids with atrophic rhinitis, which, when the former were removed, the partial atrophic condition regenerated markedly. The regeneration he observed in most of his cases of partial atrophy when he cleared away the sinus disease. He believes that the atrophic condition develops when this tenacious discharge deposits over the inferior or middle turbinated bodies; here it causes destruction of the cellular elements; the epithelium develops in marked stratification; there is a secondary contraction of the underlying connective tissue with a vascular anemia and starvation finally causing bony atrophy. He absolutely denies that the discharge usually found in the nose of atrophic cases is secreted by the turbinated body, as claimed by Zuckerkandl.

Chiari is absolutely against the assumption that sinus diseases are the cause of this affection, and brings forth the claim that atrophy occurs in children as young as two and a half years old, and one certainly cannot expect that this large amount of discharge can come from such extremely small accessory sinuses of children.

D. Braden Kyle writes very exhaustively on the subject of causes and classifies particularly from the pathological point of view. As to the sinuses being involved, he says they may be primarily or secondarily. No credit is given to the bacterial cause of this disease.

General constitutional diseases, according to this author, are responsible for many cases of atrophy, such as syphilis and tuberculosis, and atrophy is frequently associated with kidney, heart and lung diseases, causing an over-abundance of blood (passive congestion), with secondary contraction and final atrophy. He also believes in atrophic cause of atrophic rhinitis. Much stress is laid on the difference between atrophy and degeneration. The former occurs first; degeneration second.

So far, then, nothing has been said about radiograms in this disease as to the sinus involvement, and the consistency of bony structures of the turbinate and other bones of the face.

The technique of taking radiograms in this disease is the same as when we wish to determine sinus diseases. I have always made two exposures, one lateral and one anterior. The patient's face is put directly on the plate and immobilized by a compression apparatus, and he is instructed to be absolutely quiet, not even to breathe or move the lids for sixty to eighty seconds. This is accomplished by the patient signalling with his finger when he cannot hold his breath any longer; then the current is severed and the patient is again allowed to breathe, and the procedure is repeated until the full length of the exposure is made. The lateral view is exposed for from fifty to sixty seconds, and the antero-posterior for eighty seconds. The tube is placed back of the head about fourteen inches from the occiput. The plate is developed in the usual photographic manner, and after it dries it is examined by means of transilluminated light in a dark room. The plates are of diagnostic value, while the prints of them are, as a rule, very unsatisfactory, an unfortunate fact for the purpose of publication of the plates. The antero-posterior views are best for judgment of the frontal, antral and anterior ethmoidal sinuses, while the sphenoid and posterior ethmoid can only be seen in the lateral view. However, the super-imposition of the two sides makes this view not very satisfactory for diagnoses, except in connection with the physical examination.

The involvement of a sinus is judged by its density, and while I am not absolutely certain that the skiagraph is true in every instance, it is in the majority of cases that I have examined and opened. The cavities are shown as dark areas, whereas when filled with pus, thickened mucous membrane or granulations, they are shown as whitish areas, similar to the surrounding solid bone.

Of the 24 cases of atrophic rhinitis that I radiographed, I found the sinuses involved in all but 3 cases, and these were of the light type of atrophy.

TREATMENT.

Most of the authors treat the symptoms of odor and obstruction, due to the crust formation, and only the minority from the pathologic point of view. Hajek, Gruenwald, Braden Kyle and Onodi are some of the latter, and it seems their results are much better. Personally, I am of the opinion that the condition may be primary in the nose or sinuses, but that in the majority of cases, when treatment is directed towards these accessory cavities, in addition to the nasal and general treatment, the results are better. The literature

is full of cures and marked benefits from ever so many different methods of treatment, and that fact alone is enough to show that we have no specific treatment applying to all cases. The essential fact, I believe, as to the treatment is to remove the cause and then establish hyperemia, which will be followed by resolution, either partial or complete.

As to the methods of treatment that I have followed in my cases, I will report specifically in each group, for I have divided them up into six groups, so as to be able to control my experiments. The methods were:

1. Irrigation of the areas of the ostia of the various sinuses, or of the sinuses directly by means of a solution composed of iodine, formalin, and sodium chloride, associated with the suction method, as recommended by Sundermann or Brawley.

2. Operative interference, usually intranasally, by removal of the middle turbinated body, curettement of the ethmoid cell, removal of the anterior sphenoidal wall, and a large opening into the antrum of Highmore at the inferior meatus—with or without the removal of a portion of the inferior turbinated body. I do not need to say that in each case all these sinuses were so operated on, only when absolutely indicated.

3. Paraffin injections into the inferior turbinated body.

4. The use of high frequency current into the nasal and pharyngeal spaces.

5. Bier's treatment, such as constriction band around the neck, and Reti's method of compression at the posterior choanae, and Sundermann's method of constant suction.

6. Vapor therapy, by means of the Bulling's variator, using the same solution as for irrigation, only stronger.

I have come to some definite conclusions as to the value of these various methods of treatment, and will report in detail, as said before, in connection with each group of cases.

From the foregoing, it will be apparent that my treatment is principally directed to the sinuses. All other treatment is used to promote resolution or restitution of the atrophied structures by means of hyperemia and leucocytosis.

These 24 cases were divided into groups in order to be able to judge and compare the various methods of treatment, and each group of cases will be reported by taking one typical case as an example.

GROUP I. SEVEN CASES.

Case 1. Miss M., 17 years old. Father and mother and six brothers living and well. None have any trouble like the patient's. She had the usual diseases of childhood. Ever since she can remember she has had trouble in her nose, as discharging of accumulation of crusts of a foul odor. She herself cannot smell them. Suffers much with headache, and is frequently dizzy. Her throat is constantly dry and makes her cough, especially in the morning. Her appetite is remarkably diminished. Never any ear trouble.

STATUS: Poorly-nourished young woman; very anemic. Blood examination shows a lack of hemoglobin, and diminution of red blood corpuscles. Urine negative. Nothing pathological, apparently, of the gastro-intestinal tract, heart and lungs, and genito-urinary tract, or the cerebro-spinal system.

Nasal examination shows a bilateral atrophy of the inferior turbinated bodies, with apparently enlarged middle turbinated. Membrane of the whole nasal cavity of an anemic character, with minute ulcerations scattered all over the cavities. Posterior wall of pharynx and Eustachian orifices are clearly seen through the anterior nares, and appear very dry. There is no pus escaping from any of the ostia. Epi- and oro-pharynx are dry and apparently thinner than normal. Larynx and trachea negative. Ears negative.

Examination of eyes: No fundus lesion, only a hyperopic astigmatism; D. 50.

Radiogram: Shows involvement of all the sinuses, in being almost as dense as the surrounding bones. Transilluminations dull.

Puncture of the antrum revealed pus of a thick greenish color.

DIAGNOSIS: Atrophic rhinitis and pharyngitis, associated with a pansinusitis.

TREATMENT: Washing out of the sinuses by means of canulas introduced into them, or in the vicinity of the ostia, using an irrigator placed ten feet high. This fluid used was a very warm iodine-formalin-salt solution. This is followed by tilting the head forward and backward to the right and left, so facilitating the emptying of the sinuses, and then using either the Sounderman or Brawley suction apparatus, for the purpose of aspiration (Bier's treatment). Only slight improvement followed this sort of treatment, and an operation was suggested of removal of the middle turbinal, and ethmoidal, sphenoidal and antral sinus exenteration. This was accepted and done. The result was a recovery from this operative

procedure, and a marked improvement in the general condition of the nasal mucosa, marked reduction of the odor, and her general condition markedly improved. Seven weeks later a bilateral injection of paraffin (cold) was done, with a very good result of bulging of the inferior turbinated body.

Remarks. This case shows that the treatment of the sinuses was the proper procedure. The irrigation fluid of formalin, iodine and sodium chloride is one of the best, for the purpose of irrigation in this infection, of which I know, and I believe the iodine acts as a particular stimulant to healthy surfaces. The paraffin is supposed to act beneficially by reconstructing the inferior turbinal, but I believe that it also acts as a nutrient or stimulant to a healthier and better blood supply, because, as I will show in other cases, much improvement followed by simply injecting a quantity not large enough for reconstruction.

The other six cases are about the same, varying in the history and general examination somewhat, but, as a whole, required the same kind of treatment and gave about the same result. You observe that irrigation and aspiration treatment was tried before for a period of from six weeks to three months, but in all these seven cases operation was performed by the intranasal route.

GROUP II. FOUR CASES.

Case 1. Miss C., aged 22. Father died at 53; kidney trouble. Mother and three sisters well, and none have any trouble like patient's. She had the usual diseases of childhood, but not scarlet fever. Ever since she can remember she has been discharging crusts from the nose of a foul-smelling character. She can smell fairly well. Her general health is always good. Has been under treatment by a general practitioner, who directed her to wash her nose out with salt solution in the usual manner of snuffing the water, and he applied nitrate of silver, 10 per cent; also packed the nose with tampons of wood wool, without any result. Never has had any ear or throat trouble.

Status: Well-nourished young lady, and in apparently perfect health. Nasal examination showed atrophy of both inferior and turbinated bodies. The middle turbinate looked much thinner than normal, and the sphenoidal sinus, ostium maxillare and naso-frontal duct were easily probed. The general mucosa was of an unhealthy character. The post-nasal space and Eustachian orifices were plain-

ly seen through anterior nares. The mucous membrane of the epi- and oro-pharynx, as well as the larynx and trachea were normal. Ear and general examination negative.

Radiograph shows the sinuses plainly involved, but not so markedly as in Group I.

Transillumination, practically dull. Puncture of antrum negative after irrigation.

DIAGNOSIS: Atrophic rhinitis, with some involvement of a mucous type.

TREATMENT: Irrigation of the sinus with the various canulas, using the iodo-formalin-salt solution. Aspiration of the sinuses. Cold paraffin was injected into both inferior turbinated bodies, and it appeared as though the condition improved from the moment the paraffin was injected.

REMARKS: The other three cases were almost identical, and the results are the same. These four cases are all of more than one year's standing, and what I say of their improvement is from my recent observation of them. The crusts and odor are not present at any time, and the patients do not complain, except on a cold day, when they feel too much cold air rush into the throat. There is no washing or treating done at home, and only general hygienic measures are observed.

GROUP III. FOUR CASES.

Case 1. Mr. V., 25 years old. For the past two years has been under treatment of a general practitioner for a hacking cough, taking medicines without any result. His family history is good. He has been well until the above mentioned time, and does not remember of ever being very sick or having bad colds. For the past few months there have been crusts forming in his nose, and his folks complain of an odor, but he cannot smell it. His throat is very dry, and a thick crust forms in the back of his throat, that he thinks makes him cough.

EXAMINATION. General examination of heart, lungs, gastro-intestinal tract, kidneys and blood, negative.

Nasal cavity: Nostrils filled with casts of crusts. Both inferior turbinated bodies partially atrophied. The middle turbinate body is thickened and covered with a thick viscid, muco-purulent secretion. The epi- and oro-pharynx and false vocal cords are deeply inflamed, and small crusts are formed on the latter. The larynx and trachea are also injected and red. Sputum examination, nega-

tive as to tubercle bacilli, but mixed staphylococci and bacilli were found. Lots of pus cells. Suction does not reveal any pus from sinus.

Radiograph negative, except that the ethmoidal sinuses look a little cloudy.

Puncture of antrum negative.

Transillumination negative.

DIAGNOSIS: Atrophic rhinitis, with epi-, oro- and hypo-pharyngitis.

TREATMENT. Inhalation of vapor generated by the Bulling's vibrator, using a nasal tip for the nasal treatment, and mouth tip for the throat inhalation. The fluid used was the same as the irrigation fluid. Treatments are given three times a week, for ten to fifteen minutes, and each sitting or treatment continued until crusts and odor disappeared.

REMARKS. These four cases may be classed in one group, because they all give a history of late manifestations of this trouble. They are all negative, on radiographic examination. They are all practically free from headaches, and the atrophy in them all is only partial. They all got well by means of this treatment, and I only wish to say that in cases where the throat is involved, particularly the larynx, this treatment is of distinct amelioration to the patient.

GROUP IV. TWO CASES.

Case 1. Miss M., 26 years old, nurse. For several years has had trouble with the right side of her nose, crusts forming which are of a very foul odor. Her head aches on that side very much, and she cannot see so well with the eye on that side. Her family history is negative, and she only had the grippe when she was a little girl. Since then she believes this trouble began. She has been using the douche cup for a long time, but her ears get so bad when she does so that she has abandoned that. When removing the crusts has frequent nose-bleed. General examination negative, except a goiter.

STATUS: *Right Side of Nose.* Almost complete atrophy of inferior turbinate. Crusts and pus in the nose. A streak of pus going back into the throat from about middle turbinated body. This body is very large, and from the region of the hiatus semilunaris springs the characteristic Kaufman's osteophytic outgrowth so characteristic of sinus disease.

Left Nostril: Slight hypertrophies, but otherwise negative.

The throat is covered with a purulent secretion, especially post-nasally and on the right side.

Puncture of antrum. Washed out thick pus.

Transillumination: Distinctly dull on right side.

Radiograph: Very distinctly shown to be ethmoidal, sphenoidal and antral trouble in the density of the structure. The frontal appears clear.

Eye examination showed dilated pupil; paralysis of the sphincter pupillae. No fundus lesion. Vision 20-50, and does not improve.

Diagnosis: Unilateral atrophic rhinitis, associated with unilateral sinus disease.

Treatment. Complete exenteration of the antrum, ethmoid and sphenoid on right side, with the happy result of absolute relief from the headaches; the discharge and crusts less; the odor markedly diminished, and the bleeding, which, by the way, required on several occasions post-nasal packing, has entirely ceased.

Remarks. This case would certainly prove that sinus disease can and does cause atrophy. This second case is almost the same, except that the radiograph is confusing, and shows, if anything, trouble on the healthy side. I am of the opinion, however, that the fault lies in the technique.

GROUP V. THREE CASES.

Case 1. Mr. B., aged 43, laborer, single. Denies all specific history, and says he has always been fairly well, with the exception of nasal and throat catarrh. Has considerable headache. Cannot see well, and has been cross-eyed for some time—about fifteen years. For the past two years has had much trouble with his throat, being compelled to cough up small dry pieces, which require great effort. His throat and nose feel very dry at all times. Every winter, if he is exposed the least bit, he has a severe bronchitis, and once he had a pleuro-pneumonia. The crusts are not so marked of late as they formerly were. Hears poorly, and has head noises.

Status. Fairly well nourished man, whose lungs and heart are negative. Also urine and blood examinations negative.

Nasal Cavities. Double atrophy of inferior and middle turbinates. The mucous membrane of the naso-pharynx and larynx appears atrophied. The vocal cords and below them in the ventricle, and even lower into the trachea, can be seen small, dirty-looking crusts. Both tympani membrana are markedly retracted.

Transillumination: Fairly good light through the pupils.
Radiograph: All sinuses cloudy.
Puncture of antrum negative on washing out.

Diagnosis: Double atrophic rhinitis, pharyngeal and laryngeal, and tracheitis. Otitis media catarrhalis chronica.

Treatment. Bier's constricting band about the neck, low down, worn twenty-two hours out of twenty-four, and Rethi's method of compressing the veins in the post-choana by holding a perforated rubber plug in this position for twenty-four hours, changing from right to left each day.

Remarks. The other two cases are of the same character, and are treated in the same manner. The time is too short to say much about the results, as the patient has only been under treatment for about three months. However, without hesitation, I can say that the symptoms of dryness are markedly lessened, and the general appearance of the mucous membrane is certainly of a healthier character.

GROUP VI. FOUR CASES.

Case 1. Mrs. E., 35 years old; married; six children. Has for years been troubled with a dry nose and throat, and foul-smelling crusts are discharged after a great deal of effort, and soaking in salt water. The ears are very frequently stuffed after this sort of treatment. Family, childhood and adult history negative, except that she has always been very nervous.

Examination, generally, showed nothing of importance, pathologically.

Nasal fossae: Atrophy of both inferior turbinates. The upper parts of nose are covered with small crusts and a very tenacious secretion. Pharynx is dry and glossy. Tracheal membrane retracted. Hearing reduced to about one-half.

Radiographs show cloudy sinuses.

Treatment. Exclusively daily application of the high frequency current to the nasal and pharyngeal mucous membrane. The method of applying is as follows: A resonator is used for the product of the high frequency current, being supplied by an eighteen inch coil. A glass electrode extends from the resonator, which is introduced into the nose or throat, as the case may be. After the electrode is introduced, the current is turned on and is permitted

to remain at first one minute, and gradually increased to three minutes. The strength of the current is also graduated, at first light (second or third ring of the resonator), and gradually raised to the third or fourth. This treatment is carried on daily until a reaction results, something like a coryza. Then stop and wait until all irritation disappears, when the treatment is renewed, and at this time more toleration exists. It is kept up every day for one to two weeks, and then gradually every other day, etc. No other treatment is associated with it.

Remarks. All the four cases were of about the same nature, and treated in the same manner. More than two years have passed since the first case was treated by this method, and I am sure that the condition was very much improved. You understand that there are cases that I either did not want to operate on, or was not permitted to do so.

There is another diagnostic-therapeutic experiment that I would like to report in connection with this subject, namely, the use of dionin. I do not intend to go into the details of the chemistry of this substance, only saying that it is a morphine derivative. It is a sedative, but when applied to the conjunctiva causes an artificial edema of varying degree. It is used in ophthalmology very extensively as a therapeutic agent to clear up acute as well as chronic inflammatory products. I have, therefore, argued from analogy that if it acted in other parts of the body as it does in the eye, it would be of benefit in atrophic rhinitis; firstly, by causing an edema, it would show whether the connective tissue is very firmly adherent to the bone or not; and, secondly, acting as it does on the eye, it would assist in absorption of chronic inflammatory products.

I have tried it on almost all the cases, and on some normal and other conditions, with such variable results that I shall be satisfied at this time to simply put the experiment on record, and say that it acts on certain individuals and not on others. It is a toxic substance, and should be used with care, although I have never had the least trouble from its toxicity, even when injecting it into the middle ear through the membrana tympani.

Conclusions.

1. That the sinuses are very frequently involved in atrophic rhinitis, whether primarily or secondarily, is not always possible to determine.

2. That when the sinuses are involved the atrophy must have some other cause; as, for instance, bacteria, heredity, etc., for we do find so many sinus troubles with hypertrophy.

3. That radiographs are an aid in diagnosis, and should be practiced by every rhinologist.

4. That treatment directed to the sinuses is followed by improvement, much more readily than when not.

5. That surgical intervention, preferably intranasal, gives the best results.

6. That the results of local treatment, as by tamponing, massage, electricity, vapor therapy, paraffin injection, Bier's treatment, etc., are brought about by the production of hyperemia and leucocytosis, bringing about an alternating condition, and possibly a resolution or restitution of glandular structures and normal mucous membrane and even some of the erectile tissue.

PAPER:

THE IMPORTANCE OF CORRECTING PATHOLOGIC CONDITIONS OF THE NOSE AND THROAT IN PATIENTS WHO HAVE INCIPIENT TUBERCULOSIS.

WILLIS S. ANDERSON, M. D.

Pulmonary tuberculosis is a disease which attacks persons of lowered vitality. The lowered vitality results from improper or insufficient food, unsanitary surroundings, intemperance or other vicious personal habits, overwork, mental worry, and disease. Tuberculosis does not attack the strong and vigorous. The tubercle bacillus finds lodgment in tissues that are below par.

The problem to be met in the treatment of this disease is to increase the resistance of our patients. In the absence of any specific agent for this purpose, we rely mainly upon hygienic, dietetic and climatic measures.

The value of fresh air and out of door life is now generally recognized. It is customary to advise patients to live out of doors even in the coldest weather, but few physicians think of examining the

nostrils of their patients to see whether it is possible for them to obtain the benefits of fresh air by normal breathing.

A carefully regulated diet may be prescribed with digestants and stomachics to encourage the stomach to perform its functions; but if the tonsils are hypertrophied, with septic accumulations in their crypts, or if there is muco-pus in the nose or sinuses, we know that digestion will be impaired. This impaired digestion may arise from the absorption of septic material from the nose and throat, or from the swallowing of the secretions.

As an aid in the treatment of tuberculosis we should increase the bodily resistance of our patients by restoring to as near normal as possible any defective organ. The failure to appreciate the importance of this rule has lessened the chances of recovery of many tuberculous persons.

If organic defects are found after a thorough examination of the patient, judgment is required to determine what bearing they may have on the pulmonary condition and general nutrition. Organs remote from the respiratory tract, though pathologic, may have little, or no effect, on the local condition. The correction of some lesions might necessitate more risk, or a longer period of close confinement, than would be warranted in certain cases. There is such a close relation between the upper respiratory tract and the lungs, that any abnormal condition of the nose and throat should receive careful attention.

We shall study these abnormal conditions under the following heads:

Pathologic conditions of the nose, naso-pharynx and pharynx. The lesions in the nose may be grouped into two classes: First, obstruction to free nasal breathing; and second, any condition which gives rise to accumulations of mucus, or muco-pus in the nose or sinuses.

The study of the physiology of nasal breathing in the lower animals gives us many hints as to its importance in man. We know that animals that are very dependent upon nasal breathing, which if interfered with leads to serious disease or even death. Man has greater powers of adaption, therefore nasal obstruction does not produce the serious immediate effects that are noticed in the lower animals. There is difference of opinion as to what constitutes free nasal breathing. Air may be drawn with effort through the nostrils that are not free for ordinary quiet respiration. A cursory exam-

ination of the nose may show a fairly free passage, but a more careful examination may reveal narrowness at one or more points. We must remember that the breathing capacity of the nose is measured by its narrowest point. The current of air passes in a curved direction, upward and backward to enter the naso-pharynx. A comparatively small obstruction in the upper portion of the nose or naso-pharynx will interfere with the current of air in its passage to the lungs. While such an obstruction does not prevent the forcible drawing of air through the nose, it does interfere with normal, quiet breathing. Many persons claim to have free nasal breathing, when in reality they breathe all the time partially through the mouth.

If we notice in public places how persons breathe, a considerable number will be observed with the lips more or less parted; this indicates that a certain amount of respired air is entering through the mouth. The majority of these individuals do not realize that they are mouth breathers. I have asked many patients to try the experiment while walking along the street, of keeping their lips closed and noticing whether they could breathe comfortably through the nose. In many instances after walking a block, or less, they will open their mouths a little to obtain more air. After taking several breaths they can close their mouths again for a few moments. While these patients can breathe through their nose, the space is not sufficiently free to give them all the air necessary. This partial mouth breathing is not normal, and is an important factor in the etiology of the so-called catarrh of the laity. These patients are subject to colds which may be localized in the nose and throat, or involve the lower respiratory tract as well. They are frequently hoarse and have a dry irritable throat, which is more marked in the morning.

In examining the lungs of persons who have nasal obstruction, I have noticed that there is a diminution of breathing, more marked at the apices, or what is sometimes spoken of as "lazy lungs." It is not always easy for the physician to decide whether a given individual has proper breathing space or not. Several examinations may be necessary before this point can be determined. The writer suggests a certain routine in order to determine this point.

While getting the history, and without calling the attention of the patient to his breathing, notice carefully if the lips are a little separated and if some air is entering through the mouth. Ascertain

if the patient notices an alternate obstruction of the nose at night, or in damp changeable weather; if in the morning the throat is dry from mouth breathing; and if the voice lacks resonance, or has a nasal quality. An examination of the nose should then be made to estimate the amount of breathing space. After this preliminary examination the soft parts should be shrunken by the use of cocaine and a suprarenal preparation. While shrunken notice whether there are any points that are especially narrow. By examining the turbinals before and after they are contracted, an estimate can be made as to how much the size is due to intumescence and how much is due to hyperplasia, or to enlargement of the turbinated bones.

The conditions to be looked for especially are polypi, or other tumors; narrowing due to deviation of the septum; hypertrophy of the anterior or posterior ends of the inferior turbinals; and hypertrophy of the middle turbinals. The middle turbinal is often the offending member, and its importance as an etiologic factor in the production of nasal obstruction is frequently overlooked. It often causes intumescence of the inferior turbinal by interference with the circulation. In such cases it is useless to cauterize the inferior turbinal without removing the underlying cause of the intumescence. After all, it is the care and judgment of the examining physician which must finally determine the relative amount of nasal breathing.

An excessive amount of mucus, or muco-pus, may accompany nasal obstruction, and when present usually indicates involvement of one or more of the accessory sinuses. Nasal obstruction and improper drainage are the usual causes of chronic sinus involvement. Sinus disease is a menace to the health and comfort of a patient, and in itself lowers bodily resistance.

The naso-pharynx is less frequently obstructed in adults than the nose, but there remains some hypertrophy of the tissues of the vault, or a granular condition, in those cases which had lymphoid hypertrophies in childhood. This causes an accumulation of thick, tenacious mucus, which is aggravated if there is nasal obstruction. Chronic follicular pharyngitis is frequently found in tuberculous patients. It causes an irritation, with a constant desire to clear the throat, and often a dry irritable cough.

Diseased tonsils are a source of great danger to tubercular persons. There is now abundant clinical and experimental evidence

that various infections enter the body through the tonsils. Infection has been traced in animals from the tonsils, through the lymphatics and cervical glands, to the bronchial and mediastinal glands. A submerged tonsil, with diseased crypts, is capable of doing as much, or more, harm than a large, hypertrophied tonsil. Tubercular tonsils are not rare, and tubercular cervical glands are very common.

We are indebted to Dr. George B. Wood, of Philadelphia, for many facts in reference to the lymphatic drainage of the faucial tonsil, and the infection of the tonsil by tuberculosis. He uses the term, "Tonsillar lymph gland," to describe a lymph node, which is enlarged during tonsillar infection. It is situated external and slightly anterior to the internal jugular vein, and is embedded in loose areolar tissue, containing more or less fat.

Dr. Frederick A. Packard, in his Wesley M. Carpenter lecture on "Infection through the tonsils, 1900," brought together many data, which emphasized the important relation between tonsillar and rheumatic affections. Many other observers have shown that it is just as important to appreciate the role that the tonsils play in the etiology of tuberculosis. Besides the danger of direct tubercular infection, we have septic absorption from the diseased tonsillar crypts. This slow absorption often causes a slight elevation of temperature, general malaise and lowered resistance. Hypertrophied tonsils may interfere with free breathing or deglutition, and the exudate which accumulates in the crypts impairs digestion and gives a foul odor to the breath. Hypertrophy of the lingual tonsils are frequently found. The principal symptoms noticed are a dry, irritable cough, constant desire to clear the throat and congestion of the larynx.

Tubercular laryngitis complicates about one-third of the cases of pulmonary tuberculosis. A laryngitis is usually a forerunner of this condition, and imperfect nasal breathing is an important factor in the production of a chronic catarrhal laryngitis. Even where the larynx is not tubercular we commonly have a congestion and a hypersensitive condition, which in itself gives rise to an irritable cough and huskiness of the voice.

The above conditions are easily recognized by those familiar with lesions of the nose and throat, but they are not often considered in their relation to the early stage of tuberculosis. A patient with incipient tuberculosis is particularly susceptible to atmospheric

changes, because of the lowered resistance. They shudder at the thought of remaining out of doors during the winter season. If they do not breathe freely through the nose the cold, dry air is drawn into the lungs through the mouth. This causes irritation and congestion of the mucous membrane, and is a positive handicap in a patient's struggle for life and health.

We ought to realize that a case of incipient tuberculosis is not properly managed unless nasal obstructions are removed, diseased tonsils carefully dissected out, and other lesions of the nose and throat corrected as far as possible. The methods used in the treatment of these conditions are familiar to all and need not be considered in detail, although a few general points will be considered.

Fortunately local anesthesia is sufficient in nearly every case. If for any reason a general anesthetic is to be used the added danger which comes from the anesthetic itself should be considered. The danger from cocaine poisoning is so remote, if the drug is carefully used, that we can exclude it from our considerations. There is very little shock following operations upon the nose. If both nostrils are involved, the worse side can be attended to first, and a little time allowed for the patient to recuperate before the second side is corrected.

In suppurative sinusitis the cavities should be drained and washed out. The nasal route should be chosen. Several points ought to be considered before advising a serious sinus operation upon a tuberculous patient: First, does the sinus disease in itself threaten life; second, the probable amount of shock to a person already below par; third, will the after treatment be long and tedious; fourth, can the patient's symptoms be relieved by washing out of the sinus regularly, without recourse to operation. If the above points are considered very few radical sinus operations will be justified upon a tuberculous patient.

Hypertrophied lingual tonsils should be cauterized with trichlor-acetic acid, the galvano-cautery, or removed with a lingual tonsillotome according to the severity of the condition. Hypertrophy of the follicles of the posterior wall of the pharynx should be cauterized by touching the elevations with the point of the galvano-cautery, or in mild cases with trichlor-acetic acid.

In the treatment of the tuberculous we ought to consider nutrition as the ability not only to digest and to assimilate food, but also to inspire normally a sufficient amount of fresh air to meet the de-

mands of the body. As success or failure in the treatment depends upon nutrition, attention should be directed to the upper air tract to lessen air hunger. That air hunger does exist from nasal obstruction is proven by the effect upon the nutrition of children who have adenoids, by the impaired health in adults when the nasal respiration is below normal, and experimentally in animals where one or both nostrils have been closed.

Some of the symptoms noted in the more severe examples of obstruction may be ascribed fairly to pressure, and the interference with the circulation in the brain; this is accounted for by the free anastomosis between the vessels of the nose and the meninges. If this condition exists it is all the more reason why the local pressure should be removed. There is probably no difference of opinion as to the importance of the severer type of nasal obstruction, but the milder forms may be considered by some as unimportant. I feel that this is a mistake and that even the milder forms should be given careful attention.

I might cite cases to illustrate the value of the principles outlined in the above paper, but the reports would not be conclusive as the correction of the lesions of the nose and throat has been supplemented in every case by careful hygienic, dietetic and medicinal treatment. I feel convinced from my own experience that the correction of abnormal conditions of the nose and throat aids materially to the chances of cure in tuberculous patients.

PAPER:

FURTHER STUDIES IN NASAL THERAPEUTICS.

JOHN A. THOMPSON, M.D.

A former paper of mine on the same topic called forth the statement from a colleague that all were agreed on the treatment of nasal inflammations. He said the patient was operated on for the removal of part of the turbinated bodies and recovered without any further care after the operation wound had healed. Should this statement really represent the views of most rhinologists, I am compelled to assume the role Goethe gives to Mephistopholes, "the

Spirit that contradicts." While there are many diseases in the nose requiring operative intervention, there are others where surgical treatment can only do harm. Acute rhinitis untreated lasts from ten days to three weeks. Any additional irritation by operation when the membrane is already inflamed can only increase the suffering of the patient and prolong the attack. Its duration may be lessened to four days by intelligent local treatment. A gain of from six to seventeen days of good health is certainly worth an effort.

Purulent rhinitis in children is common. It results in the formation of no new tissue and causes no permanent obstruction to nasal respiration. Free the nose thoroughly from the accumulated secretions and the child breathes normally. It certainly would require great surgical enthusiasm to find indications for operation in these cases. They can be quickly and easily cured by local medicinal treatment.

Atrophic rhinitis is a destructive process from the beginning. To aid the wasting process by cutting out tissue already being destroyed does not seem to be good treatment. To stimulate the circulation of the parts and improve their nutrition so the atrophy may be checked is far better for the patient. This does not in any way interfere with the surgery of the complications that may be present. The drainage of infected sinuses or the removal of degenerated turbinal tissues does not supplant the treatment of the causal disease. It can be checked, and its disagreeable symptoms relieved without resorting to surgery.

More than one-half of all cases of hay fever can be relieved by local measures so they can continue their work in comfort. The hypertrophies or new growths accompanying hay fever frequently require removal in the interval, but operation during the attack can only result disastrously. To save the majority of these patients the interruption to business and the expense entailed by an annual trip to some region where the disease does not prevail is a therapeutic result worth striving for.

In membranous or croupous rhinitis any injury to the nasal mucosa results in an extension of the false membrane. Even forcible removal of this morbid product is injurious. In this disease medicinal treatment is alone to be considered.

In la grippe as in acute rhinitis the severe inflammation of the membranes of the nose forbids operative work. Our study should

be how we can best and most quickly relieve the local inflammation in addition to the treatment of the general symptoms.

Simple chronic rhinitis produces no obstructive lesions in the nose, and operative treatment meets none of the pathological conditions.

The prevalence of diseases of the nose which are best treated medicinally is to me a sufficient reason for resuming a study which, while not as exciting as the technique of sinus operations, will be of benefit to a much larger number of patients.

In my former paper I presented a study of cleaning washes and of the therapeutic action of menthol, camphor, thymol, eucalyptus, resorcin, the aromatic oils, nitrate of silver, iodine, carbolic acid and monochlorphenol. Next in value to these is boric acid. It is the least irritating of the effective antiseptics, and can be used in the nose mixed with equal parts of an indifferent powder. Probably the best combination is with stearate of zinc. It is a protective and mild sedative in its local action. It checks the secretion of the mucous glands where it is applied. Where the secretion becomes crusted the combination of borax and boric acid in an ointment will dissolve the crusts and prevent decomposition of the secretions. This combination is especially valuable in eczema of the vestibule and in purulent rhinitis. Eczema in the vestibule of the nose, where the lining is a delicate skin, not a mucous membrane, is very common. Crusts form where the epithelium is diseased or destroyed and their removal is painful and attended by troublesome hemorrhage. Applying an ointment containing a drachm each of borax and boric acid in an ounce of vasaline to the diseased areas softens the crusts so they can be removed without pain or bleeding. In mild cases the use of this remedy alone will result in a cure.

The purulent rhinitis of children can be cured in from four to six weeks by the same remedy used liberally in the nose at bedtime. The salve is of such consistency that it melts slowly at the temperature of the body, and with the child lying down runs back over all the diseased membrane, on which it exerts its healing influence. In acute rhinitis where the desquamation of the epithelium increases the sensitiveness of the nose, insufflations of a powder containing equal parts of stearate of zinc and boric acid gives great relief by protecting the sensitive mucosa and preventing the growth of bacteria in the secretions. The familiar purulent discharges of the third stage of a "cold" will be greatly lessened in duration and amount if this powder is used.

In hay fever there is an additional reason for its use besides its action as a protective to inflamed and sensitive membranes. Dunbar has shown that the irritating portion of the pollen that causes hay fever is soluble only in alkaline solutions. If we can keep the nasal mucous mildly acid it will not dissolve the pollen and there will be no irritation. This can be partially accomplished by spraying the nose with a solution of suprarenal extract. When the swelling is lessened by this remedy the powder of stearate of zinc and boric acid can be inflated and the patient assured several hours of comfort.

Stearate of zinc is rapidly winning a high place as a protective in general surgery. Whether or not it has any other than a mechanical action is not yet proven. As a protective it is very useful in the second stage of acute rhinitis and in hay fever.

Bismuth subgallate was first introduced to the medical profession as a dusting powder to be used in place of iodoform. Its value as an astringent and antiseptic in inflammations of the bowels has diverted attention from its use on operation wounds in the nose. We cannot do aseptic work in the nose. Where asepsis cannot be maintained the next best treatment is free drainage and antiseptic applications to the wounds. Any kind of dressing in the nose so interferes with drainage that after danger of secondary hemorrhage is passed nothing but a dusting powder can be used with comfort and safety. While the opinion is based purely on clinical observation, I believe there is less suppuration and more rapid healing after operations with bismuth subgallate than with any other powder.

The use of the various preparations of the suprarenal gland in operative work in the nose is too well known to need repetition. The therapeutic use of this animal extract in other conditions has been given but little attention. In the early stages of hypertrophic rhinitis the condition present is a congestion due to lack of vasomotor tension. The infiltration with leucocytes and the production of connective tissue follow this congestion, which Kyle has described as intumescent rhinitis. If we can cure the beginning hypertrophic rhinitis without operation we can leave the patient a normal turbinated body, one capable of performing its physiological function. Suprarenal preparations stimulate the contractile muscles of the blood-vessels. They overcome temporarily the pathological dilatation. I know of no demonstration quite so startling

to the student eager for operative work as to show him one of these cases with both nostrils full of apparently solid tissue, and then to show him the same case five minutes later, with open nostrils, after spraying with a suprarenal solution. The demonstration that the obstruction is congestive, not organized tissue, is perfect. We do not amputate for chronic congestion anywhere else in the body without trying therapeutic measures. There is no sound reason for the prevalent craze for doing so in the nose. The stimulant action of the suprarenal gland on the dilated vessels is rarely followed by a paralytic dilatation. It can be repeated with the same tonic effect as massage or electrical stimulation would have. Properly used, it will cure many cases that have heretofore been considered operative. Menthol and camphor combined in an oily solution, not strong enough to be irritating, have a similar though lesser effect, and can properly be given to the patient himself to use as an adjuvant. Combined with resorcin, the suprarenal preparations will abort an acute rhinitis. In hay fever no other local remedy gives so much relief. In acute inflammations of the accessory sinuses the suprarenal preparations, by contracting the nasal tissues and permitting the escape of the retained secretions, relieve pain better than morphine. In a few cases the preparations are irritating, producing the symptoms of acute rhinitis and cannot be used. The great majority are benefited by them.

Cocaine is used in the nose for operative work. It is not a remedy that can be safely given for chronic diseases. It aggravates the local disease when regularly employed, and there is great danger of inducing the cocaine habit. In acute sinus inflammations, particularly ethmoiditis, the excruciating pain may be relieved by spraying the nose with a suprarenal solution and then applying a pledget of cotton saturated with a 10 per cent solution of cocaine over the normal opening of the sinus. The local application to the inflamed nerve endings gives more relief than a hypodermic injection of morphine. The application must be made by the physician himself and the patient should not be told what remedy is used. Should the acute attack be the beginning of a chronic inflammation, he may be tempted later to use the remedy himself if he knows what it is.

The other local anesthetics now on the market are similar in their action to cocaine, except that they do not produce a local anemia, and are consequently less serviceable than cocaine.

When a long-continued analgesic effort is desired either orthoform or anesthesin may be employed. Neither one is active when employed on the unbroken mucous surface, so their field of usefulness is limited. Orthoform causes sloughing of the ulcerated surface to which it is applied, so it should be used only on tertiary syphilitic ulcers in their early painful stages.

The vegetable astringents have been replaced by more efficient agents in the treatment of nasal diseases. Their chief use in rhinology is as hæmostatics. Gallic and tannic acids combined in the familiar Mackenzie styptic solution are the most effective. A little known and little used remedy that is very valuable is powdered kino. Where a slow, persistent oozing occurs from either a wound in the nose or from a diseased membrane, repeated insufflations of this powder will check it when other remedies fail.

The new silver salts have won golden opinions from the oculists for their efficiency in inflammations of the conjunctiva. In the nose they are apparently useless. I have never seen any noticeable benefit from their use, and those of my colleagues to whom I have spoken have expressed the same opinion.

Hydrogen peroxide is an active antiseptic, a stimulant to the circulation and a solvent of crusted secretions. These qualities make it valuable as a cleansing agent in atrophic rhinitis, tertiary syphilitic ulcers in the nose and after operations on the accessory sinuses. It may be used indefinitely in atrophic rhinitis, but must be discontinued in other conditions as soon as repair of the ulcer or wound begins. It will destroy the new granulation tissue as readily as it does pus, and prevent healing if used at the wrong time. It has also been highly recommended as a haemostatic.

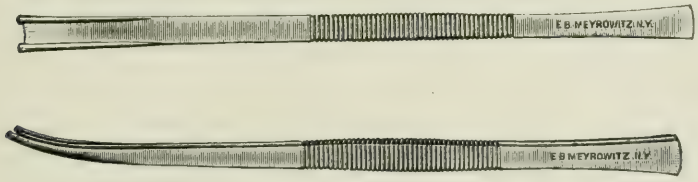
The consideration of other remedies must be postponed to a future occasion.

PAPER:

TWO CHISELS DESIGNED FOR THE PREVENTION OF INJURY TO THE FACIAL NERVE IN THE RADICAL MASTOID OPERATION.

JOHN J. KYLE, M. D.

I desire to call the attention of the Society to two chisels designed for the prevention of injury to the facial nerve in the radical mastoid operation. As a rule, injury to the facial nerve results from the too forcible breaking down of the arch of the posterior osseous wall covering externally the aditus ad antrum. The chisel is so designed that two blunt edges absolutely prevent the cutting surfaces entering the inner walls of the middle ear, unless driven with inexcusable force.



The chisel is also of practical value in removing the external wall of the zygomatic process, when the Rongeur cannot be used with ease.

One chisel is straight, designed for removing superior portions of the wall, and the other is curved, designed for removing the inferior wall.

I have used these chisels a number of times and have been well pleased with them. The operator can use less care in removing the posterior wall than with the ordinary chisel. From experience I believe I am justified in recommending this chisel to my confreres.

The width of the chisel varies from 6 mm. to 8 mm.

PAPER:

PRIMARY MELANO- SARCOMA OF THE NOSE. REPORT OF A CASE AND REVIEW OF THE LITERATURE.

CORNELIUS G. COAKLEY, M.D.

The infrequent occurence of this rare variety of Sarcoma, developing primarily in the nasal mucous membrane, makes my case worthy of recording.

Mrs. R. P., Italian, aged 58 years, was referred to me January 4th, 1907, by Dr. Sebastian Saladino, of this city. The family history is negative as to the occurrence of any tumor, benign or malignant, so far as the patient is aware. Her father died of bronchitis (?) and her mother during childbirth. At the age of 25 she married and bore two children, both of whom died in infancy, one of whooping cough and the other of diphtheria. Her first husband having died she married her present one 21 years ago. She had been a robust, healthy woman all her life until the latter part of 1904, when she noticed obstruction to respiration in the left nasal cavity. The obstruction continued to increase and about the middle of January, 1905, she consulted her family physician, Dr. Dominic Saladino, who on examining the left nasal cavity found a large mass which he took to be a polyp. He removed the growth with a cold wire snare and remarked upon its very black color. The nasal obstruction was somewhat relieved, but the discharge from the left naris, sometimes of bright arterial blood and other times of inky blackness, greatly alarmed her.

About December 1st, 1905, she became the patient of Dr. Spicer of this city, to whom I am greatly indebted for an account of the condition as he found it at that time. The left nasal cavity was almost completely occluded by a dark colored mass which filled the posterior two-thirds of the cavity and projected into the naso-pharynx. The exact point of attachment of the bulk of the mass could not be determined. There was also a rounded, black mass on the left side of the septum about half an inch posterior to the

vestibule and a little above the floor of the nose. It was elevated at least one-eighth of an inch above the mucous membrane and the line of demarcation between that and the healthy membrane was very distinct. A similar black growth was observed on the outer wall of the left nasal cavity, about the middle of the vertical height of the vestibule and at the junction of the skin and mucous membrane portions of the nose. The left inferior turbinate was also involved along its lower border with a deposit of the same black growth. At this time the patient was complaining of some headache and frequent epistaxis. Dr. Spicer removed a mass of tissue about the size of a walnut and considerably relieved the nasal obstruction, but the hemorrhages continued. No microscopical examination of the tissue was made. Small pieces of black growth were several times removed until September, 1906, when Dr. Spicer took his vacation.

The patient then consulted another laryngologist in our city, who treated her until about December 1st, 1906, removing portions of the growth. During this time, however, nasal obstruction was not relieved and the right nasal cavity also became occluded.

At the time of her first visit to me both nasal cavities were filled with a dark, ropy, grumous mass, which made inspection of them impossible. After syringing away some of this material and contracting the tissues as thoroughly as possible with cocaine and adrenalin, there could be seen on the left side of the septum, near the floor, a sharply outlined black mass, half an inch in diameter and in the position as described by Dr. Spicer. It projected a quarter of an inch above the general level of the mucous membrane. A neoplasm was also found on the outer wall of the vestibule as he described it, and so much of the lower border of the inferior turbinate as was visible, namely, about a quarter of an inch, was similarly infiltrated with a black mass. The rest of the naris was completely filled from roof to floor with a nodular, black mass which bled upon the slightest touch with a cotton tipped applicator. An unusual condition was noticed, namely, that any portion of the mass that was touched with cotton, stained the latter as if dipped in black ink.

On contracting the mucous membrane of the left nasal cavity the septum was found to be deflected slightly into that naris and there was extensive hypertrophy of the mucous membrane of the

inferior and middle turbinates, so that these were practically in contact with the septum.

The cotton tipped applicator could be passed beneath the inferior and middle turbinates and between them and the septum without any resistance, until the posterior snares was reached, when a soft, spongy mass could be felt which stained the cotton as did the growth in the left nasal cavity. Thorough examination of the right naris failed to detect any involvement of its mucosa by the neoplasm, or any undue pigmentation of the mucous membrane.

No post-rhinoscopic examination could be made on account of the soft palate hanging tightly against the posterior pharyngeal wall, as a result of long continued, total mouth breathing. The growth on the left side of the septum and the neighboring adjacent mucous membrane was cocainized. With a right angled knife, such as is used for submucous resection, an incision was made through the healthy mucous membrane at a distance of an eighth of an inch from the circumference of the neoplasm. By carrying the incision down through the periosteum to the cartilage and bone, as is done in submucous resection, I was enabled to lift and remove this island of new growth, surrounded by healthy tissue, with a periosteal elevator. The cartilage and bone beneath the periosteum was perfectly healthy. The deepest layers of the periosteum had evidently not been invaded by the neoplasm. The tissue was placed immediately in 4 per cent formalin solution and sent to Dr. Harlow Brooks for microscopical examination. The following is the report of the pathologist:

New York University.—The University and Bellevue Hospital Medical College, 26th Street and First Avenue, New York City. Department of Pathology.

January 10th, 1907.

My Dear Dr. Coakley:—

Microscopical examination of the pigmented nasal tumor sent me a few days since shows the mass of the growth to be sharply differentiated from the nasal mucosa. The tumor has apparently original from the connective tissue layer beneath the mucous membrane and is composed, except for its vessels, entirely of connective tissue elements. The tissue is composed of a mass of polymorphic connective tissue cells, mostly of spindle shape and of great varia-

tion in size; some of these cells are very large. All are distinctly of the connective tissue variety.

A good many of the cells enclose within them considerable masses of brown and black pigment.

Blood vessels are abundantly present and the walls of a good many of these channels are incomplete and atypical, made up in some instances of the cells of the new growth.

As regards the shape of the cells, the growth should be classed as a Mixed Cell Sarcoma, but on account of the very large amount of pigment present it should be arranged as a Melano-Sarcoma. It is a tumor highly malignant in character and one likely to set up early and widely disseminated metastases.

Very sincerely,

HARLOW BROOKS.

On January 11th the patient came to my office and a portion of the growth, situated on the middle meatus, was removed with a snare and embedded in formalin gelatine in order to preserve its color and gross appearances. On January 25th the area of the septum, from which the growth had been removed at the first visit, was completely healed. In view of the long standing history of the case, and the good results that other operators have reported from curettage and the use of the galvano cautery, the patient was advised to submit to this method of treatment. This was declined. The patient was given a powder containing powdered suprarenal gland and antipyrin, grs. V of each, simply to see what effect it would have in controlling the hemorrhages.

Patient claims that there has been less hemorrhage since using the powder. On February 8th the patient came to the office and a third piece of the mass in the left middle meatus was removed with a cold wire snare. Moderate bleeding followed, controlled by nasal tampon.

My friend, Dr. John McCoy, to whom I am greatly indebted, has made a most complete search of the literature and the following is an abstract of the cases we can find recorded:

CASE I. Vienois. (*Lyon Med.*, 1873, No. 18.)

Woman aged 63, operated on by Ollier's method, for a growth which had given rise to distortion of the external nose, with

exophthalmos and intense supraorbital pain. Patient succumbed eight days later of pyemia.

Case II. Osio. (*Rev. de Cien. Med.,* Barcel. 1875.)

Man aged 55, in which tumor had apparently existed for eight months. It had its origin in the outer wall of the right nasal passage, and subsequently invaded successively the opposite side, the antrum of Highmore, the orbit, and the sphenoidal sinuses, giving rise to deformity of the external nose, nasal stenosis, pain, exophthalmos, and injection of conjunctiva. A radical operation was attempted, followed at the end of seven days by death of the patient from meningo-encephalitis.

Case III. Burckhardt. (Bericht über die Chir. Abtheilung des Ludwigs-Spitals Charlottenhilfe im Jahre, 1884.)

Woman aged 44, suffered from nasal stenosis, severe pain and frequent hemorrhages. The growth involved both nasal cavities. Access to the growth was obtained by slitting the nose and the growth was removed by means of a sharp spoon and the finger. The subjective symptoms were improved for a short time, but there was a recurrence five months after the operation.

The above three cases were taken from Bosworth's "Diseases of the Nose and Throat."

Case IV. Lincoln. (*New York Med. Journ.,* 1885.)

Mrs. L., first seen in November, 1884. Twenty-eight months previously the patient first noticed obstruction in the right nasal cavity and at the same time bloodstained discharge from that nostril. Dr. Agnew of Philadelphia removed a growth from this nostril in April, 1883 (it was said to be cancerous). Two months later there was a recurrence and a second operation and a third recurrence and operation in November of that year. In February, 1884, the patient went under the care of a physician in Rome, N. Y., who burnt out the nose with plasters and paste, but without giving the patient any relief. Dr. Lincoln found the right nasal cavity completely filled with a plum-colored mass which bled on the slightest touch. The growth could be seen in the posterior nares. On November 18th, under ether narcosis, a galvanic cautery loop was passed around the growth, which was cut out and the base cauterized. The patient was seen repeatedly until October, 1885, and there was no recurrence. Dr. F. Ferguson, pathologist, reported the growth to be a Melano-Sarcoma; prognosis *grave.*

Case V. Shalcross, I. G. (*Hahnemann's Medical Monthly*, January, 1892.)

German woman aged 72, was first seen in August, 1891. She came to the Clinic with a diagnosis of polyp, following grip. She complained of obstruction in the left nasal cavity. About April of that year a physician removed a polyp (?) with scissors. Severe hemorrhage lasting two hours followed the operation. Four weeks later the operation was repeated because of recurrence of the nasal obstruction. The hemorrhage was again very profuse. It was found that a blue-black growth filled the left naris and protruded through the vestibule. There was considerable bleeding from this nostril and occasionally fetor to the discharge. The growth was attached to the triangular cartilage of the septum by a broad pedicle. It was also adherent to the middle and inferior turbinates. On August 15th, the bulk of the mass was removed with a snare and the base curetted with a sharp spoon. Profuse bleeding followed, which was controlled by a nasal tampon. August 22nd, more curettage followed by application of chromic acid. Microscopical examination of the growth showed it to be a round celled Melano-Sarcoma. As the woman was becoming progressively weaker no further operation was performed. The outcome of the case has not been recorded.

Case VI. Heymann, P. (Melanosark. d. Nasenh. Naturforschervers. Köln 1888 and Halle 1891.)

Fifty year old man had symptoms of nasal polypi. These had been removed a number of times from the right naris before he came under Heymann's care. Examination revealed the left naris obstructed by a tumor of a dirty gray-black color, the surface superficially ulcerated, of hard consistency and situated on the septum to which it was attached by a broad base. The tumor was cut off with a snare and the base curetted. Fourteen months later the patient was still healthy and free from any recurrence of the growth. The extirpated tumor was the size of a walnut, covered with thick mucous and on section was gray-black in color. Microscopical examination revealed a Melanotic Sarcoma, composed of round and spindle cells, alternating in alveolar and fibrous arrangement. The pigmentation was various. Alongside of very black particles were found brown and entirely unpigmented areas. The patient was examined every three months without showing any signs of return of the growth until the spring of 1891, when on the same side of

the septum, in the neighborhood of the former scar, but separated from it by a centimeter of healthy mucous membrane, was a growth about the size of a hazelnut. This was removed in the same manner as the previous tumor. Microscopical examination revealed the same picture as in the former instance. At the same time Heymann found in the other nasal cavity a polypoid hypertrophy of the anterior end of the middle turbinate. This, upon microscopical section, showed the usual structure of such a growth, and in addition, enormous melanotic cells of irregular form and varying size, scattered throughout the tissue. These were evidently cells which had wandered into the tissue and not a part of the structure of the tissue. Near by, however, were found pigmented masses and crystals not embedded in cells. These pigmented portions were peculiarly glistening and evidently arose from previous hemorrhages.

Case VII. Güder. (Melanosarcome de la fosse nasale droite. Geneve, 1894.) Quoted by Schrott, Inaugural-Dissertation, Pseudomelanosarkom der Nasenmuschel, 1897.

Patient, 47 years old, had observed a small growth in his right nasal cavity for the past two years. Frequently complained of coryza and epistaxis. The tumor gradually increased in size until the patient applied to physician for help. The tumor was removed, but rapidly recurred and the patient went to the Clinic. The right nasal cavity was found to be filled with a tumor the size of a small grape, reaching as far forward as the vestibule of the nose. He was operated on in June, 1879. Microscopical examination of the growth revealed a Melano-Sarcoma. Up to the time of reporting, 1894, there was no recurrence.

Case VIII. Schrott (*loc. cit.*) also reports a case from the *Arch. f. klin. Chir.*, 1887, which I have been unable to verify.

Three years before the patient had a fall with an injury to the nose. He attributed the nasal obstruction to this. Curettage of the mucous membrane was followed in two months by a recurrence. Next followed partial resection of the superior maxilla, but the growth returned in the maxillary sinus. Then the superior maxilla was resected and the patient died.

Case IX. Kafemann. Quoted by Schrott (*loc. cit.*)

A 58-year-old woman complained of nasal obstruction and profuse epistaxis for ten days before coming for treatment. Examination revealed the left turbinate greatly swollen and the middle

meatus was entirely obstructed by a black growth which bled on the slightest touch. In no place was the bone exposed. The right nasal cavity was absolutely normal and transillumination showed the maxillary sinuses free. Extirpation of the tumor was accomplished by means of a galvano cautery snare and the base thoroughly destroyed with a thermo-cautery. About two months later a recurrence of the growth was observed at the junction of the vestibule and the nasal mucous membrane. For the purpose of microscopical examination a portion of this mass was removed. The examination revealed a Melano-Sarcoma. Many of the cells contained melanotic pigment. Six days later there was swelling on the left side of the nose. This was removed by a radical operation; the wound healed primarily. The patient so far is free from any recurrence. (1897.)

Case X. Michael. (Melanosark. d. Nase. Internat. Med. Congr. zu Berlin. Laryngol. Section, 1890.)

Fifty-year-old patient complained of left-sided nasal obstruction for a year. Inspection of the nose showed polypoid masses, which were darker than ordinary polypi, with black streaks through them. After removing these the posterior portion of the nose could be seen filled with a dark blue-black mass. This was removed and the base curetted and destroyed. A year and a half later there was no recurrence. Microscopical examination showed a Mixed and Round Cell Sarcoma, with a small amount of connective tissue framework. The scattered pigment was partly in the spindle cells, partly in the round cells and partly in the stroma.

Case XI. Michelsohn, P., Königsberg. (Reported by Heymann, *loc. cit.*)

A case of Melano-Sarcoma involving the inferior turbinate. Jacob, Inaugural-Dissertation, Das Melanosarkom der Nase, Leipzig, 1900, reports that he was unable to learn anything more about this case.

Case XII. Bristow, A. T. (*Brooklyn Med. Journ.*, 1900.)

In August, 1899, the patient was first seen complaining of left nasal obstruction and epistaxis for the past year. A black mass completely filled the left nasal cavity, involving a small portion of the left side of the septum and the inferior turbinate. The patient was etherized and the nose thoroughly curetted. When partial healing took place it was seen that there still remained a small portion of the growth on the septum. Three months later this growth

on the septum was curetted away in the same manner. Microscopical examination showed an Alveolar Sarcoma with such an amount of pigment as to be considered a Melano-Sarcoma. No recurrence for 20 months.

Case XIII. Kummel, W. (Die bösartigen Geschwülste der Nase. *Heymann's Handb. der Laryngol. u. Rhinol.*, Bd. III., S. 878.)

Patient came under his care at the Strassburg Ear Clinic some years ago. What first appeared to be an ordinary polypoid tumor of the nose, was on microscopical examination, after removal, pronounced a Sarcoma. The abundant gold-yellow pigment evidently resulted from previous hemorrhages. The patient was transferred to the Surgical Clinic, where he was operated on. The superior maxilla was resected. He died not long after from general melanosarcomatosis, with typical melanin deposits.

Case XIV. Brieger. (Jacob, *loc. cit.*)

Eighty-year-old man who previously had Melano-Sarcoma of the eye, which was operated upon. For some months past he had nasal obstruction and for some weeks past had blood tinged, foul smelling discharge from nose and nasal pharynx. By posterior rhinoscopy there was found a black tumor attached to the inferior turbinate and invading the lateral wall of the choana and nasal pharynx. Excision was accomplished by means of a snare. The microscopical examination was as follows:

"The structure of the polyp was a round cell Sarcoma. Throughout the entire tissue there was a scattering of pigment, mostly in the interstitial tissue, especially in the neighborhood of the blood vessels. Only occasionally were pigment granules found within the large sarcoma cells."

Case XV. Brieger. (*loc. cit.*)

A 70-year-old man came to the office with the announcement that he had on that same day looked into his mouth and seen a black growth in the back part of it. By simple inspection of the mouth I saw behind the velum, somewhat to the left of the uvula, an entirely black mass about the size of a hazelnut. Further examination revealed that the entire naso-pharynx was filled with this black mass, which was very soft on palpation. On inquiring of the patient it was found that for the past few weeks he had complete nasal obstruction and for the past few days epistaxis and a ringing in the

left ear. Examination of the ear showed marked retraction of the membrana tympani on both sides. With the exception of some swollen lymphatic glands there was nowhere else on the body any evidence of swelling or metastasis. For the purpose of microscopical diagnosis a piece about the size of a walnut was removed through the mouth, hardened in alcohol and sections made and examined. Low power magnification of the non-stained sections showed dirty brown, large and small specks scattered irregularly through the field. High power appearance showed these to be resolved into a powdered, crystalline mass, and elsewhere irregularly shaped homogeneous masses. They were mostly situated within the cells. The cells of the growth were large and round with large nuclei and separated from each other by fibres of connective tissue, here and there arranged in groups like uvula. The pigment which was deposited in the interspaces was stained and all the pigment in the new growth cells was diffusely black. On account of the diagnosis, the appearance of the swollen lymphatic glands indicating metastasis, the great age and weak condition of the patient, the radical cure by means of an operation was not considered. Only whenever the patient desired were walnut sized pieces of the growth removed through the mouth by means of a snare.

Both patients died a relatively short time after the first examintion.

Case XVI. Cozzolino, V. (*Arch. f. Laryngol. u. Rhinol.,* Bd. XV.)

Man, 58 years old, seen on March 8th, 1899. Complained of obstruction in right nasal cavity for several months, and occasionally neuralgic pains in frontal and orbital regions. There was also a black, purulent discharge from the right naris. Examination of the right naris showed a blackish, bean-shaped, nodular growth which filled three-fourths of the cavity. On posterior rhinoscopy a simiiar growth was found on the border of the choana in the region of the inferior turbinate and a portion of the middle turbinate. With scissors and a cold wire snare the growth was extirpated with the usual amount of hemorrhage. The following day the base of the growth was burned out with a thermo-cautery. A month later the patient complained of a return of the growth, which was not surprising as Dr. F. D'Orlando diagnosed the sections made from the original mass as Melano-Sarcoma. On re-examination of the patient it was found that the growth had invaded the wall of the maxil-

lary sinus. The patient was taken to the hospital where the late Prof. C. Gallozzi resected the superior maxilla. About two months after this recurrence took place in the skin, which up to that time had been healthy, and invaded the orbital cavity. Death followed from general sarcomatosis.

A study of these cases shows practically an equal distribution as to the sex—7 males, 6 females, and four in whom the sex is not mentioned. As to age, the youngest was 44 and the oldest 80; the average, in the 12 cases in which the ages are given, is 59.

As to the situation, a very large proportion seemed to originate in the structures of the outer wall of the nares; two only are recorded as involving the septum alone. In two other cases, in which there were multiple foci, some nodules were found on the septum, near the floor. It is interesting to know that the olfactory region of the septum has not been so prone to this type of sarcoma as the other portions of the nasal chambers.

Unfortunately, the histories of many of the cases are incomplete as to the duration of the disease prior to treatment. In case No. IV. it was 2 1-3 years; No. VII., two years; No. VIII., possibly three years; No. IX., only ten days; No. X., one year; No. XII., one year; No. XIV., some months; No. XVI., several months; and in my case, 2 1-3 years. This would seem to show that in many of these cases the growth was very slow. If we take into consideration the slow progress of the disease in my case, since she was first seen by Dr. D. Saladino, it is more than probable that the first invasion of the nose took place many months before the neoplasm had grown so large as to obstruct the naris and cause her to seek relief from this inconvenience.

The mortality rate as derived from the recorded cases is probably misleading and it is greatly to be regretted that the various authors have not made supplementary reports on their cases after a lapse of some years following the various operative procedures.

Of the 17 cases, *seven,* Nos. I, II, VIII, XIII, XIV, XV, XVI, are recorded as having died. Of these, *five* cases, Nos. I, II, VIII, XIII, XVI were subjected to extensive operations, such as resection of the superior maxilla. Judging from the reported condition of cases Nos. III and V, they probably did not live very long. Case No. VII had no recurrence after 15 years, No. V, none after 1½ years; No. VI recurred slightly after three years and was re-operated on; No. IX recurred in two months, was operated on and

"some months" later was free from return. No. X, no recurrence after 1½ years; No. XII, slight recurrence after three months, re-operated on and no recurrence at the end of a year and a half.

All of these cases reported as non-recurring were treated by curettage, galvano-cautery snare, thermo-cautery, or some combination of these therapeutic measures. This has so impressed me that, should my patient consent to allow me to attempt a cure, I will adopt them in preference to more radical surgical procedures.

DISCUSSIONS.

DR. RICHARDSON: I recently had a case of melanotic sarcoma of the nose in a child four years old. The growth seemed to involve the outer walls of the nose and was operated upon. Six months later the pharynx became involved and the child died. A microscopical examination showed it to be very much like the specimen exhibited by Dr. Coakley.

DR. BAKER: I removed a malanotic sarcoma of the optic nerve fifteen years ago. Five years after the removal it recurred in the liver and other organs and the patient died.

PAPER:

HYPERTROPHY OF THE PHARYNGEAL LYMPHATIC RING AS A CAUSATIVE FACTOR IN THE PRODUCTION OF EPILEPTIC EQUIVALENTS.

WALTER S. DALY, M. D.

As far back as history shows and tradition indicates epilepsy has afflicted man. Methods without number, drugs beyond estimate, have been employed for its cure. Yet epilepsy is still with us, and still no specific has yet been found. In view of this fact, the great importance of preventing epilepsy becomes apparent, and to prevent it in one group of persons we must be able to recognize a tendency before it eventuates in a habit, and in another realize the enormous importance of certain peripheral irritations as a cause.

A child of neurotic heredity is especially prone to motor nerve storms, and the first indication of an epileptic tendency is often an

attack of infantile convulsions. It is not assumed that such an outbreak invariably precedes epilepsy, but it is unquestionably the fact that a large majority of epileptics of whom an accurate history is obtainable, have had these convulsions in infancy.

Furthermore epilepsy from the viewpoint of modern neurology, is not necessarily characterized by convulsions, for, it is an indisputable fact that an attack of epilepsy may occur without a convulsive seizure, the convulsion being substituted by certain mental disturbances called epileptic equivalents.

Such attacks in childhood exhibit a morbid sensibility to peripheral irritations and should be esteemed a peremptory indication for years of watchful care, our aim being to prevent the occurrence of other convulsive seizures and the formation of a habit of convulsion or epilepsy.

An epileptic equivalent therefore is a certain state of consciousness differing not only from normal consciousness, but from the state of consciousness that usually occurs in epileptic attacks. The term really signifies a psychical state that in some degree takes the place of an epileptic seizure.

These phenomena present many points of interest, are of rarer occurrence than true epilepsy and are more difficult to diagnose.

Harry P., aged 10, was a farmer lad, whose family history was negative and whose relatives on both sides were long lived and healthy.

Personal History—Patient developed naturally as a baby, but at the age of two years had what was called a "Worm fit," which came on quite suddenly with nausea and vomiting, followed by an attack of what was undoubtedly a seizure of the grandmal type of epilepsy presenting the classical symptoms, general tonic and clonic spasms, cyanosis and loss of consciousness.

Following the attack, which lasted five minutes, the patient urinated very freely. During the next three years the child averaged about three of these attacks a year; the attacks, however, gradually increased in severity and duration. At the age of five the attacks suddenly ceased, to be followed by a period of quiescence, lasting five years, during which, the boy, to all appearances, acted like other boys, was very quick to learn, and showed no abnormal traits.

Suddenly during the night of February 18, 1906, the patient was taken with a spell, during which he complained of being afraid,

wanted to leave the house, cried out spasmodically, putting his hands to the right side of his head, as if his ear, on that side, were aching. When the pain was at its height, the boy appeared dazed and aimlessly moved about the house. This spell, of insane manifestations, lasted nine days, then gradually subsided and for a period of two weeks the boy was apparently as well as ever.

He then had a second attack which came on suddenly while in the barn helping his father remove a calf from one stall to another. The first thing noticed was that he began asking his father where his calf was; he apparently did not comprehend his surroundings and had forgotten what he had been doing just a few minutes before; with expression vacant and ideas confused he wandered from one person to another, finding nothing to please him, nothing to pacify him.

Shortly afterwards, he complained of pain, now in the left side of his head and frequently cried out: "Oh, my ear!" This condition lasted for a period of seven days and then ended abruptly when the patient cried out: "There it has gone." After three or four days of sluggish mentality, he gradually brightened up and appeared well for about two weeks, at which time he was visiting his aunt, who noticed, about the noon hour, that he began to talk strangely, to sing and to fear being left alone. At two o'clock he complained of severe pain in both sides of the head, accompanied by the usual insane manifestations of the previous attacks.

The attack lasted nine days, gradually subsiding in three or four days, to be followed by a fourth attack two weeks later.

The fourth attack suddenly began about nine o'clock in the morning when the boy rushed out into the road and looking up the hill nearby, began calling: "Hello, Frank," and shook his hand over his head. There was no one in sight—evidently hallucinations of vision. In a few hours pain in the head supervened and he appeared dazed and confused. He did not seem to recognize the presence of his parents, even though they were by his side; would frequently call his mother, even while she was trying to quiet him. Evidently illusions of identity.

During all these attacks, there was entire oblivion on the part of the patient, of all that had happened.

It was during this last attack, and on account of some possible mastoid implication, that the writer first saw the case with Dr.

George Young of Prescott, Ontario, the family physician, and to whom I am indebted for much of the early history. On my arrival at the house, the boy, crazed and delirious, ran away, and it was with difficulty that he was apprehended. A thorough examination was impossible; so, the parents were advised to bring the boy to my office when a lucid interval would occur, which I had every reason to believe would come in about ten days.

About two weeks afterwards, on June second, he was given a thorough examination with the following findings: The patient is a well developed boy of ten years, light complexion, light hair, lips slightly thickened and gaping, mouth breather, with a lack of resonance of the voice, obliteration of the normal lines of expression of the face, pupils moderately and equally dilated, the special senses normal and unimpaired, nothing abnormal about the ears or mastoids, a course tremor of the tongue and fingers, the other parts of the body being steady. The patient has control of the various muscles of the body, showing good strength; nothing abnormal was found in the chest and urinary organs; temperature and pulse normal, the roof of the mouth is narrow and highly arched. Examination of the throat showed marked hypertrophy of the pharyngeal lymphatic ring. Finding nothing, beyond an hypertrophy of the tonsillar ring, to account in a causative way, for the several attacks of which the boy had been the victim during the past four months, I called in consultation, Dr. R. H. Hutchings, Psychiater and Superintendent of St. Lawrence State Hospital at Ogdensburg, N. Y., who, after careful inquiry, diagnosed the attacks as Epileptic Equivalents. It was brought out that these epileptic manifestations, in a boy epileptically inclined, might be produced by some peripheral irritation, such as, in this case, hypertrophy of the pharyngeal lymphatic ring.

So accordingly on June fourth, I removed the hypertrophied condition of the tonsillar ring, with the result that all the epileptic manifestations have disappeared and have remained so to the present day.

In view of the striking results here obtained, it must be concluded that this case of Epileptic Equivalent was either wholly or largely due to the irritation in connection with the hypertrophied tonsillar ring. As would be expected, his mental condition has also improved very much since the removal of this peripheral irritation.

The case, from early childhood was evidently one of epilepsy, the manifestations of which were caused by some peripheral irritation, which, if removed, would, in all probability, safeguard the child against future and more serious attacks.

Any irritation, whether it be in the alimentary canal, worms, carious teeth, stomach affections; in the respiratory organs, polypi, adenoids, tonsillar hypertrophies; or in the eye, ear or sexual organs may precipitate an explosion in a patient epileptically inclined. It is probable that the peripheral irritation in some way interferes with or disturbs the circulation of blood through the brain. There seems to be first a spasm of the cerebral arteries causing a profound anemia of the brain, and later a dilatation of the cerebral vessels causing a congestion or hyperemia of the brain.

Up to within a few years, because the symptom-complex of certain types of minor epilepsy, occurring in early childhood, did not bring out prominently those classic symptoms of epilepsy: tonic, or clonic convulsions with loss of consciousness, the medical profession has passed them by, entirely overlooking and ignoring their importance and gravity. Allow incipient manifestations such as: "Worm-fits," "teething fits," "stomach fits," "lapses," "faints," "night horrors," "sleep walking," "absent mindedness," and other apparently innocent phenomena, which are nothing more than Epileptic Equivalents, to pass unnoticed and uncared for, and you may have thoughtlessly launched your little patient upon the full tide of epilepsy.

If there is one disease in which early recognition is desirable, it is epilepsy. The early recognition of these obscure epileptic manifestations of early life, which present themselves under so many and so diverse forms is really a difficult problem. For unless we are constantly on the lookout for them, we are very apt to view them rather as the innocent mannerisms of childhood life than the true landmarks of a disease so full of torment for the body and so disastrous to the mind.

Epileptic Equivalents in childhood commence so insiduously that they are hardly noticed at first. Look at the little child of two or three years drumming away on its plate at meal time, and, all of a sudden, stops—strikes some peculiar dazed attitude for a short time and then resumes its play with a smile. At first the parents regard these spells as cute, but when the child reaches the age of

five or six, they think it is high time for the child to discard its baby cuteness; so, in the effort to correct these hitherto cute, but now, annoying ways, the parents resort to all kinds of discipline and even punishment.

The convulsive tendency of a child in whose life we find such mother-named spells as "worm-fits," "teething fits," "stomach fits," "faints," "lapses," or any other convulsive manifestations of similar nature should be a matter of solicitude to the attending physician as long as that child lives. It should be an invariable rule to search thoroughly every such individual for points of irritation. Wounds of the head or other portions of the body, astigmatism and other imperfections of the eyes, diseases or malformation of the throat and nasal cavity, carious teeth and retained milk teeth, aural disease, adherent prepuce or other irritation of the genital organs are among the irritations which have in very many cases provoked epileptic manifestations.

The importance of thorough examination is increased by the fact that the reflex epilepsy may engender the epileptic habit.

Even with the passing of infancy there still remain certain critical periods such as the first and second dentition and at puberty, during which that same convulsive tendency of former days must be guarded against. Epilepsy, in its development, occurs with greatest frequency between the ages of ten and sixteen.

"The keynote to the whole situation, as Sprattling says in his article on 'Unrecognized Epilepsy,' is, in mistaking apparently innocent phenomena for phenomena of the gravest import. Muscular contractions and distortions are nothing in the prognosis of epilepsy compared to the disastrous form of the disease that may be so silent in its appearance, course and termination as wholly to escape unskilled observation. We are just beginning to learn a great truth in the treatment of epilepsy, similar to the one it took us hundreds of years to learn about tuberculosis, and that is, that freedom from the disease is often but little more than a matter of right living."

It is not my intention to convey the impression that this young boy was cured of his epileptic manifestations, by the simple removal of his hypertrophied tonsillar ring, rather, that another blow at the mentality of this epileptically inclined boy was warded off.

For as it has been well said: "The convulsive tendency of an individual must be respected so long as that individual lives." But I do make a plea for the early recognition and proper treatment of Epileptic Equivalents, of whatever nature, as they occur in the practices of the fellows of this society; so that hand in hand, with the neurologists of to-day, no obscure nervous disease due to a peripheral irritation whether it be in the eye, ear, nose or throat, may be allowed to pass unrecognized and uncared for.

BIBLIOGRAPHY.

1. Bullette. The Laryngoscope, 1906.
2. Spratling. *Jour. Am. M. A.*, 1906.
3. Ch. Fere. La courbature comme, Equivalent Epileptique, *Revue de medicine*, 1903.
4. Bianchini. *Revue Neurologique*, 1904.
5. Janet. *Boston Med. and Sur. Jour.*, 1905.
6. Fletcher. *Med. and Surg. Monitor*, 1903.
7. Gomers. *British Med. Jour.*, 1906.
8. Ingbert. *The Alienist and Neurologist*, 1906.
9. Turner. *Lancet* (London), 1905.
10. Ashley. *Lancet* (London), 1905.
11. Shirres. *Vermont Med. Monthly*, 1905.
12. Tuke. *Psychological Medicine*, Vol. II.
13. Gordon. (*Jour. Am. Med. Assoc.*), 1907.

MEETING OF SOUTHERN SECTION, HELD IN LOUISVILLE, KY., FEBRUARY 22, 1907.

WHEN SHALL WE ADVISE TYPMANO-MASTOID EXENTERATION, IN THE TREATMENT OF SUPPURATIVE OTITIS MEDIA, AND IN WHAT PERCENTAGE OF CASES MAY WE EXPECT A CURE.

H. O. REIK, M.D.

During recent years the prognosis of suppurative middle ear disease, including all of its complications and eventuations, has very materially improved. A better realization of the importance of the disease has led to its earlier and more persistent treatment, and antiseptic surgery has enabled the aurist to save the lives of many persons whose conditions would, a few years since, have been looked upon as hopeless. Of perhaps more importance is the fact that surgical improvements have put it in the power of the aurist to prevent many of the serious complications of this disease.

A purulent inflammation of the middle ear may persist for many years without causing serious disturbance of health or destruction of any tissue beyond the confines of the tympanic cavity. On the other hand, it is becoming generally recognized now that the mere existence of such a purulent focus is a constant menace to the health and life of the individual bearing it, and that at any moment it may flare up into an active process which will be extremely difficult to check or control.

I suppose it may be taken for granted that this Association would recommend the persistent treatment of every case of purulent otitis media until cured, employing the simplest measures possible as long as they give promise of effective results, but resorting to surgical intervention whenever required to overcome an obstinate otorrhœa. Without at all exaggerating the possibility of dangerous complications or sequellae ensuing from direct extension of sup-

puration, from the tympanum to neighboring vital structures, we are safe in saying that no person who is the subject of a purulent discharge from the middle ear should be permitted to look upon his condition as one without danger to life. Aside from the risk of the graver consequences attending purulent invasion of the mastoid process, the soft tissues of the neck or the cranial cavity, we must remember that the victim of chronic otorrhoea is suffering a steady drain upon his supply of vitality and that in addition to this fixed charge against his resources he carries an undeterminable, yet constant, risk of secondary personal infection and an incubator for bacteria to supply contamination to his neighbors.

It becomes our duty, therefore, to teach the family physician and the public to recognize the fact that purulent otorrhoea is a symptom of a disease which has a most important bearing upon the patient's continued enjoyment of health and life; that it demands immediate and continuous treatment until a cure results, inasmuch as it is susceptible to cure in almost every instance; and, that while we sometimes speak of the measure which is employed as a last resort in the treatment as "a radical operation," it is not *radicalism* but *conservatism* which demands surgical intervention in chronic suppurative otitis media when other means of treatment have failed of effect.

The prognosis of suppurative otitis media, when properly treated, is extremely good. The majority of all acute cases respond promptly to proper local medicinal measures. By such treatment is meant simply the procuring of free drainage of excretions from the tympanum and the maintenance of as perfect cleanliness of that cavity as is possible. But, in spite of the early institution of skillful treatment, some cases appear to be, in the very nature of things, destined to pursue a chronic course. This tendency to chronicity may be the result of excessive activity on the part of the infective agent, of inability on the part of the patient to respond to reparative processes, or, of peculiar anatomical conditions or pathological alterations which favor the extension or persistence of the necrosis. General hygienic care and tonic treatment supported by patient, well-directed and persevering efforts to sterilize the tympanum will render the invading foe innocuous and bring about a cure in the large majority of this class of cases.

Neglect is perhaps the greatest factor in the causation of chronic purulent otitis media. Ignorance of the character and dangers of

the disease, reliance upon home treatment, carelessness in the employment of remedies and measures properly recommended by the physician, or the use of agents better fitted to excite than to allay purulent inflammation are among the explanations we frequently have to ascribe for the existence of a chronic otorrhoea when first it comes under our notice. But even these chronic cases have a very favorable prognosis when brought under proper treatment, for it has been demonstrated that taking them as they come, and including cases of many years' standing, fully one-half will recover under simple drainage and antisepsis.

Something less than 50 per cent, then, of the chronic suppurative otitis medias will require the consideration of some more energetic measures, such as surgical assistance for the removal of polypoid tissue or necrotic ossicles through the external auditory canal, or, the more extensive surgical intervention embodied in the so-called radical operation. Ossiculectomy, intratympanic curettage, occlusion of the tympano-pharyngeal tube by a flap from the tympanic membrane, and removal of the tympanic scute through the external canal are simpler surgical measures which promise satisfactory results in some cases. It is surprising what a large percentage of the very worst-looking cases will respond satisfactorily to the exhaustive employment of painstaking measures to cleanse the tympanum of debris and free it of infective material. If due care is exercised in diagnosing the factors accountable for chronicity in each case and conscientious efforts made to remove or overcome them, efforts which shall be continued patiently and perseveringly over a period of several weeks or months if necessary, according to the circumstances attending the particular case, I believe but a small minority of even the chronic otorrhoeas will require the operation of tympano-mastoid exenteration. It seems to me this operation is properly to be held in reserve as a last resort for those cases which do not succumb to milder forms of treatment, but that while an honest effort to procure healing by simple measures should precede consideration of surgical attention, indefinite prolongation of such treatment is not justifiable, and the fact that serious complications have not supervened does not excuse interminable delay. Not all cases are cured even by the radical operation, but the vast majority are, and so, only an insignificant percentage of the whole number of otorrhoeas is left unhealed after thorough treatment. That this small proportion will steadily decrease in the future as

the technique of the major operation improves in the hands of the mass of operators, is to be expected.

Nothing has been said regarding those cases of either acute or chronic otitis media in which positive evidences of mastoid or cranial invasion are present; it being assumed that all are agreed upon the necessity for prompt operative intervention as the only safe form of treatment and that the operation of complete tympano-mastoid exenteration should be performed if necessary in order to ablate the disease. It is the object of this paper to consider more particularly that large group of chronic suppurative otitis medias in which the predominant symptom is otorrhoea and in which no serious complications have arisen. Concerning these cases, if we admit the fairness of the prognosis stated above and accept, as a foundation for determining the treatment to be selected, the belief that while most cases can be cured by drainage, antisepsis and middle ear operations a small number will require the major operation, and that this operation is not only a justifiable procedure but should, as a rule, be employed rather than permit the disease to continue, it becomes necessary to consider the following questions: 1. What are the indications that justify tympano-mastoid exenteration? 2. What are the dangers and risks of the operation as compared with the disease untreated? 3. What may we safely say to the patient regarding relief from the otorrhoea and the restoration of function?

1. What are the indications for tympano-mastoid exenteration in the treatment of chronic suppurative otitis media, without evidences of mastoid or cerebral complications?

Purulent otorrhoea is, perhaps, the single symptom complained of, and, assuming now that other forms of treatment have been properly tried and have failed to control this discharge, we must be guided by a study of the excretion, the determination of the pathological lesion within the tympanum, and, the patient's general physical condition. Ascertaining the nature of the bacteria present in the pus affords very little, if any, assistance, but such investigation should always be made. As a rule the infection is a mixed one. When streptococci are present in considerable numbers we may bear in mind the especially virulent character of this organism, but it is not safe in any given case to look upon the diplococci or staphylococci as less dangerous. I have seen a fatal result in one case of pure infection by the staphylococcus albus, an organism

that is looked upon as comparatively innocent. Of much more importance is the discovery of bone dust or epithelial cells in the excretion. If the washings from the tympanic cavity be centrifugalized and the sediment, under microscopic examination, shows particles of bone structure, we have positive evidence of bone destruction going on and this is one of the strongest reasons that can be urged for operation. Likewise, the finding of epithelial cells in the pus or in the washings warns us of the possible presence or formation of cholesteatomatous masses, and, if in addition cholesterin crystals are discovered, we have positive assurance of the existence of a dangerous element. These features of the microscopic investigation are the most important single elements in determining the advisability of surgical intervention.

As regards the gross pathological changes in the tympanum, very little information of prognostic value can be gained from their inspection. Lesions of the internal wall can rarely be made out, because the disease, when it does spread in that direction, most commonly passes through the round window or, less frequently, through the oval window after erosion of the foot-plate of the stapes. Pus exuding from the oval window or from the promontory, particularly if it rapidly reappears after being wiped away, is positively diagnostic of labyrinthine invasion and the appearance of granulation tissue springing from the inner tympanic wall, with roughened carious bone underneath, is characteristic of necrosis of the labyrinthine capsule. Tinnitus and vertigo accompanying purulent otitis media, with or without the objective evidences of labyrinthine disease just referred to, while not affording conclusive proof of the extension of purulency, are sufficiently suggestive to justify an exploratory operation, and this should always be in the nature of a complete tympano-mastoid exenteration. When dealing with large polyps which protrude into the external auditory canal we may well consider the advisability of attempting their removal by a simple snaring operation, to be followed by appropriate local treatment, or, the performance at once of the radical operation. Such growths almost invariably spring from an area of carious bone, the extent of which disease can not be prejudged. Forcible evulsion has not infrequently been followed by serious complications; the tearing away of a sequestrum opening up healthy tissue to the pyogenic organisms, which seem to become remarkably active after perhaps a long period of lethargy. If it appears that the polyp has its

attachment to the roof of the tympanum or its internal wall, it is probably wiser either to make its removal part of an exenteration operation, or to inform the patient that such an operation may soon become necessary. I am, personally, inclined to look upon a middle ear polyp whose base I can not accurately decide to be situated in a comparatively innocent site as sufficient reason for recommending the radical operation. When a polyp is removed in this way the operation can be completed satisfactorily, the entire suppurative process probably be disposed of at the same time, and, the operator has better control of the situation for the prevention of future complications.

Another visible evidence of extra-tympanic purulency is the finding of pus trickling down over the internal wall from the region of the aditus ad antrum. This affords a strong inference of antral infection and as irrigation is presumed, herein, to have failed, proper drainage can be secured only by operation through the mastoid. In those cases of suppurative otitis media in which the ossicles are still present, though possibly diseased, we have naturally to consider the relative merits of middle ear operations through the canal and of complete exenteration. It is scarcely within the province of this paper to discuss that matter, though some mention of it must be made. It was understood as a premise that whenever clearly applicable simple middle ear operations would be given preference over the more extensive major procedure. Naturally there will arise a series of cases wherein opinions would differ as to the applicability of the several operations. I can imagine one surgeon advocating radical measures while another of equal ability and professional prominence would in the same case prefer to try first, at least, ossiculectomy. It is conceivable also that the latter might secure a cure in some cases. Consequently there is room for much honest difference of opinion in the selection of cases. The conservative ground to occupy would seem to be, a preference for the radical operation only when granulomata, epithelial cells or bone dust in the pus, or clinical symptoms of extension of the disease beyond the confines of the tympanic cavity, point to the necessity for some operation more thorough than can be performed through the canal.

Occasionally the patient's general state of health may be a determining factor in the decision to operate. Just how much influence a chronic purulent otitis may be exerting upon the general

system it is always difficult to say, but it is reasonable to suppose that sufficient toxines may be absorbed from the tympanic disease to prove a seriously disturbing obstacle to the growth and development of a child or to cause a decline in the vitality of an adult. It has seemed to me that this possibility has too seldom received its need of consideration. When considering the indications for operation I should be, therefore, more strongly inclined towards intervention if the patient's general physical condition was below par than in a robust person, other things being equal.

2. We come, then, naturally, to the second subdivision of our topic, the prognosis of the operation itself. What are the possible dangers of the operation? How do these risks compare with those of the neglected disease?

Under the head of prognosis we should consider: (a) the question of mortality; (b) deformity of the face; (c) failure to cure the condition for which the operation is performed; (d) further impairment of the hearing.

(a) Any operation which requires the preliminary institution of anesthesia is, of course, attended by the possible mortality of the particular anesthetic employed. As anesthetics are given today in the best hospitals, this danger is reduced to the lowest calculable point and is so small as to be practicably a negligable quantity; though the patient or his guardians should always be informed that death from anesthesia is a possibility. In view of the fact that it is generally impossible prior to operation to ascertain with any degree of accuracy the extent of the necrosis, and recognizing also the fact that some of the most careful clinical observers have disclosed at operation complicating lesions which were entirely unsuspected, we must admit the possibility of some few fatal results from the excitation of latent purulent foci or from the coincidence of having operated at the moment when an otitis media was breaking into vital structures. But has the operation any mortality *per se?* Theoretically, one would be inclined to answer this question in the negative, but, practically, it seems impossible to do so. The region attacked is one where a slight error in technic may be followed by serious consequences and few of us will claim infallibility. A very small number of cases, yet sufficient to demand consideration, have been reported in which death followed the operation without explainable cause. At the 1904 meeting of the American Otological Society, Jack reported a death after operation, for the relief

of otorrhoea, upon a particularly robust and healthy man, and Harris spoke of a similar instance occurring in the practice of Dr. Toeplitz. The fact that but few fatal results have found their way into otologic literature does not prove their infrequency, but there is no apparent reason for assuming, as some have, that a sufficient number of fatalities have occurred to give the operation an appreciable mortality.

Zeroni, in a most interesting and instructive article on Post-operative Meningitis (*Arch. f. Ohrenhlk.* Dec. 15, 1905, p. 199), reports a critical analysis of 40 cases of death following this operation for the cure of otorrhoea, which he has been able to find in German literature. Careful post-mortem examination showed that in 29 there had existed an unsuspected labrinthine disease, in 10 there was a latent meningitis or extradural abscess, and, one remained unexplained. This argues the necessity for a more careful search for these deep-seated conditions in cases which we propose to submit to operation. He believes the operation less dangerous than the disease, because a study of these autopsy materials convinced him that most of the cases were steadily progressing to a serious termination and would have inevitably ended fatally had no operation been performed.

(b) » Since almost all operators now seek primary closure of the mastoid wound in this operation we may pass any consideration of the deformity from post-aural openings. The only deformity of an annoying character that may result is that of facial paralysis. Even the most skillful operator, taking every precaution to protect the nerve, will occasionally have a paralysis following this operation. When such an unfortunate sequel results we should be slow to impute carelessness or clumsiness to the surgeon. The mere exposure of the nerve sheath to the atmosphere, to infection, or to pressure from the dressings may cause a neuritis or a paresis, and I believe that is often the explanation of those cases in which appearance of the paralysis is delayed for from twenty-four to forty-eight hours after the operation. Touching the nerve with the instruments is, of course, the most probable cause of the majority of these accidents, but unless the nerve is grossly injured the chances of recovery are good. Complete severance of the nerve or gouging out a portion of it may result in permanent loss of function. The simpler traumatisms or irritations produce only temporary loss of power. In spite of the belief in a good prognosis, one neverthe-

less views a post-operative facial paralysis with distress and apprehension.

(c) Complete cure of the suppurative otitis media does not always result from a satisfactory exenteration. With the best and most experienced operators there still remains a high percentage of failures to cure otorrhoea by this operation. Satisfactory statistics are difficult to arrive at, partly on account of the varying basis on which the indications for the operation are placed. Thus the surgeon who employs the operation for nearly every case of chronic suppurative otitis may be expected to show a higher percentage of cures than one who performs exenteration only in those cases that have been first subjected to every other form of treatment without avail. It may be said also that so far there have been very few statistics published from which conclusions might be drawn. Some of the most prominent surgeons making such reports, and who probably operate only when other forms of treatment would not promise good results, are the following: Stacke has reported 37 cases operated upon with 27 cures, (73 per cent); Panse 57 cases with 31 cures (54 per cent); Grunert 43 cases and 24 cures (55 per cent); Poli 40 cases with 32 cures, (80 per cent); and, I recently heard Dr. Dench, in a discussion of this subject, say that while in the first hundred cases he had only about 70 per cent of successes, in a second series of cases about 80 per cent of cures had resulted.

(d) The effect upon the hearing, is a question invariably asked by the patient, but I am not sure as to how much consideration it deserves. Conservation of the hearing or of a fractional portion of it has a different value for different individuals. A great many people might be deprived entirely of the function of one ear without being seriously handicapped in their business or social relations. On the other hand, persons whose condition demands acute hearing power can ill afford to lose any or, having lost some by disease, must avoid risk of its further diminution; particularly is this true when both ears are involved. We have all seen cases of extensive destruction of the membrane and ossicles, with persistent otorrhoea, in which the hearing remained nearly normal. In such a case, unless there is some urgent reason for operating it is quite proper to estimate the possibilities of further impairing the function of the organ. In the attitude I have taken, of recommending operation only in those cases of extensive middle ear dis-

ease in which other treatment has failed, the hearing power is usually a negligible quantity and occupies a minor place in the consideration of operative treatment.

A few operators have claimed remarkable improvement of hearing following operation in some cases, but as nearly as I can estimate it we are not, as a rule, justified in promising or expecting improvement in the hearing and in many cases will find that it is impaired by the operation. Should we be so fortunate as to eradicate the disease and secure a healthy skin lining for the new cavity formed by the junction of the antrum, tympanum and external auditory canal, it is quite probable that in the majority of instances this very process of epidermization, which contributes to the success of the operation, adds something to the impairment of motion of the stapes. The better the mobility of that ossicle prior to operation the more likely is it to suffer from this cause. It is perhaps only in those cases where the stapes is firmly fixed by fibrous tissue that the operation improves its ability to respond to sound waves, by liberation from the restricting bands.

How do the risks of the operation compare with those of the disease? It is impossible to make a satisfactory comparison in as much as accurate statistics are not obtainable on either side. I have stated my belief that the operation is not a very dangerous one to life and that most of the deaths that have followed the operation have occurred from some complication, previously unsuspected, rather than as a direct result thereof. Neither can the dangers of the disease be stated explicitly in figures. We all know, however, that the majority of purulent affections of the brain have their origin in neglected otitis media and that many cases of infective enteritis and fatal broncho-pneumonias, especially in children, originate from the same source. A most significant fact to be remembered in this connection is that no reputable life insurance company will take a risk upon an individual who is or has recently been the subject of a purulent otorrhoea. The business man's estimate of such risks is worthy of consideration, at least it may have more weight with the lay mind than any medical argument. In a very instructive paper by Whitehead[1] on the complications of temporal bone disease, based upon a study of the work performed at the General Infirmary at Leeds, England, during the last fifteen years, I find the following significant state-

1. *Archives of Otology*, 1906, xxxv., No. 5.

ment: "There was an antecedent history of chronic otorrhoea in 21 out of the 33 cases of meningitis; in 17 out of the 20 cases of sinus thrombosis; in 20 out of 21 cases of cerebral abscess; in 13 out of the 15 cases of cerebellar abscess, and in 1 out of the 2 cases of combined cerebral and cerebellar abscess." This comes from a general hospital, where patients were not admitted primarily because of ear disease.

In conclusion, viewing the question from these various standpoints, I would formulate the following rules for guidance when considering the treatment of persistent chronic suppurative otitis media.

1. Broadly speaking, practically every case of suppurative otitis media is assumed to be susceptible of cure by one means or another.

2. Every case of chronic suppurative otitis media, without symptoms of intracranial invasion, should be treated patiently and persistently for a reasonable length of time, but not indefinitely, by well directed efforts at cleanliness and antisepsis through the external auditory canal. When it becomes evident that these simple measures or minor operations cannot cure the disease, tympano-mastoid exenteration should be advised unless in a given case there exists some special reason of a socio-economic character that justifies delay and the risks of the disease.

3. The clinical evidences of an inveterate purulenccy that may help one to decide the question of when to recommend operation are, the finding of cholesteatomatous masses, epithelial cells or bone dust in the washings from the middle ear, the tracing of the source of pus to the mastoid antrum or labyrinthine capsule, or the existence of granulomata springing from carious areas of the tympanic wall which can not be directly inspected and treated.

4. The possible dangers of the operation are believed to be far less than those of the disease.

5. The patient should be told that not every case is curable, even by an operation, (the percentage of cures in the obstinately chronic cases probably approximating 70 per cent), that the hearing power will probably not be improved and may be somewhat impaired, but, that the serious nature of his disease warrants surgical intervention as a prophylactic measure.

6. Every patient upon whom an operation of tympano-mastoid exenteration is contemplated should be most carefully studied, for

a considerable period of time, in order that the slightest evidence of latent meningitis or purulent labyrinthitis may be detected, and when there exists any reason to suppose that the disease has extended beyond the bounds of the tympanic cavity the patient or his guardians should be told that an element of danger attends the operation. The possibility of post-operative complications can not be ignored and the surgeon must safeguard himself.

PAPER:

TREATMENT OF ATROPHIC RHINITIS WITH STRONG SOLUTIONS OF NITRATE OF SILVER WITH MASSAGE.

GEORGE F. KEIPER, A. M., M. D.

That the proper treatment of atrophic rhinitis is one of the great problems yet unsolved by the rhinologist goes without question. In fact, Dr. George L. Richards, in a paper before the Section of Laryngology and Otology of the American Medical Association, last summer, at Boston, characterizes "Atrophic Rhinitis a Reproach to Rhinology." Further, the multiplicity of remedies proposed and after a trial discarded, is evidence also for the statement. We seem to be far from a proper solution of the therapeutics demanded.

In this brief paper, I bring you my experience after fourteen years of practice.

At the outset we must recognize, if possible, the cause of atrophic rhinitis, and ask ourselves, What is atrophic rhinitis?

In this discussion, we will purposely omit such causes as syphilis and confine ourselves to its local treatment, recognizing in all cases where needed the value of internal medication to meet general conditions involved. Moreover, we will not use the word "cure" unless qualified by the word "symptomatic"; that is, it may be impossible to restore the nostrils of the sufferer to their former normal condition and function. We will be content to rid the patient of the dreadful fetor and crusts. If we can cause reproduction of bone and mucous membrane, we will count both as so much more gained

than we had hoped. It is easy to tear down, but it is quite a different problem to build up structures in the human body.

Its etiology may give us some clue to its treatment:

Three bacterial formations have been found in these cases, namely, the Pneumococcus, Cocco-bacillus foetidus of Pertz, and the Bacillus mucosus of Abel. These bacteria are resistant to ordinary antiseptics in such strength as we are accustomed to use them.

In the August number for 1899 of the Annals of Otology, Rhinology and Laryngology is an article which covers thirty pages by Professor V. Cozzolino, of Naples, who discourses extensively and learnedly therein upon "The Bacteriology and Histology of Ozena." He concludes thus:

"My conclusion is that the bacillus mucosus is the etiologic factor in the production of two of the most disagreeable *symptoms* of ozena, viz.: fetidity and crusts; but it is by no means to be classed as the specific etiologic agent of ozena, as I maintained in the discussion of the bacteriologic etiology of ozena at the International Congress of Otology, in Basle in 1884. The etiology of the bone atrophy, and, in consequence, of the mucosa, also, can be found in the nutritive alteration of the tissues of the turbinate bodies or of one turbinate body. This change begins in the bone and is often associated with a congenital, general, systemic disorder. I wish to emphasize the fact that the ozenous patient is born *ozenous;* that is to say, the child, which afterwards suffers with the full manifestations of the ozenous affection, comes into the world with a special predisposition for these nutritive changes, which determine as erosion of the bone and its ultimate destruction and atrophy of the mucosa of the turbinate bodies."

We have, then, two etiologic factors: (1) the bacterial, and (2) the hereditary predisposition.

First, let us examine the work of Cozzolino from the bacterial side. In all forty-two cases which he examined for bacteria he found the bacillus mucosus. However, in his experiments relative to germicides preventing its growth, the strongest solution of nitrate of silver used was 2%, and with no results. In fact, with the long list of antiseptics used, only four proved efficient, viz.: trichloracetic acid, 100%; trichloride of iodine and corrosive sublimate in from ½ to 1% solutions, and creosote, 4%. He seems to think that a solution of nitrate of silver stronger than 2% is not tolerated by the nasal mucous membrane. As oculists, we know that in gon-

orrheal conjunctivitis of the adult, or the ophthalmia of the newborn, we sometimes use as high as 4% solutions of nitrate of silver to the everted conjunctival surfaces of the eyelids, and with good results.

Attracted by the work of Abel in 1893 and the advice of Meyjes, as given in the second edition of McBride (1894), the writer began the use of very strong solutions of nitrate of silver to the nasal mucous membrane, beginning with a 12% solution and carrying the treatment up to a 30% solution. However, instead of simply applying it to the nasal mucous membrane, as Meyjes recommends. the writer proceeded *to rub it vigorously into the nasal mucous membrane as far as possible, at the same time exerting a tapping motion.* In other words, the general principles of massage are applied to a special field.

This brings us to the second point in etiology, Hereditary Predisposition. Whether it be true or not, one thing is certain, the bone and overlying mucous membranes atrophy. The query is pertinent—can these structures be encouraged to grow? Massage has demonstrated its value in other organs of the body in producing growth, and why not in the nose. Hence, proceeding on this assumption, the writer has combined two treatments into one, namely, massaging nitrate of silver into the nasal mucosa. The organic silver salts have been tried and found wanting. In all cases a few applications suffice to cause the odor to disappear and the mucous membrane to take on a more healthy aspect, due, no doubt, in a great measure to the disappearance of the crusts and the stimulation which the silver solution, plus massage, produces. And often are we gratified to find the nose fill up with a regenerated turbinal and overlying mucosa.

The method of procedure is as follows: The nasal mucous membrane is thoroughly cleansed of all crusts and the best solution to use in the atomizer is the warm normal saline solution. It may be necessary to dislodge some by the cotton tipped applicator and some may need to be picked out with the ordinary ear forceps. Then a twenty-five per cent solution of cocaine is carefully applied to the mucous membrane with a cotton tipped application. This is done but once. After waiting awhile for anaesthesia a 1:1000 solution of adrenalin chloride is likewise applied if a tendency to bleeding exists. The adrenalin is not absolutely necessary, as the nitrate of silver is hemostatic. The nose is then dried. The 12% solution of nitrate

of silver is then applied on the cotton tipped applicator by rubbing and vibratory massage by hand. There is no particular advantage in the machine invented for that purpose. Nature has better equipped us. Then the nose is sprayed with the camphor menthol solution given below. The patient is given directions as to how to cleanse the nose with the warm normal saline solution, which is poured into the nose either by a tablespoonful or the little glass douche, or with the Dr. Vilbiss irrigator, introducing the little tube well into the nostrils. Every hour during the day time he or she sprays the nose with the following.

℞ Mentholis .....
Camphorae aa, ...gr. XXX
Misce et adde.....
Olei Eucalypti .....gtt. XXX
Glymolis .....℥IV

The patient returns to the office every other day until the fetor has disappeared and then twice a week, and as time progresses the solution is made stronger until a 30% solution is reached.

PAPER:

PRIMARY SYPHILIS OF LIPS AND TONSILS.

A. R. THRASHER, A. M., M. D.

I beg to call your attention to a few cases of innocently acquired syphilis by way of reminding you that all such cases are not readily recognized and again to impress on your minds the fact that mucous patches on the lips and tongue are exceedingly virulent agents in conveying the contagion of syphilis. My attention has been repeatedly called to this by cases unmistakable in character that had been overlooked by the family physician and in some instances even by a specialist in cutaneous and throat diseases.

We are very apt to accept as correct a diagnosis given us by a well known physician, especially when we know him to be an intelligent and experienced practitioner. Secondary syphilis of the mouth or throat is apt to be recognized, although when the case is out of the ordinary it is at times mistaken for some other affec-

tion. Only recently there came to my office a "german garden" of syphilitic ulcers of the pharynx which had been treated by a dermatologist of more than local celebrity as tubercular, and was called by another lupus until forced to the proper diagnosis by seeing the results of treatment.

The following cases which I have the honor to report are in no way unique, but will serve to again bring before you the importance of this trite subject. In a record of two thousand cases of primary syphilis Bulkley reports five per cent as being extra genital and by far the largest portion of these occurred in the mouth. These cases occurring usually in innocent parties where no thoughts of impure lives could be entertained renders it all the more incumbent on us to early recognize them and to give them prompt relief.

Case I. Mrs. B., aged forty, was brought to me by her family physician who was also a cousin of her husband. She had an obstinate sore throat which the doctor had thought was a follicular tonsillitis, but as she was making no headway he sought a consultation. The right tonsil was slightly enlarged and hard to the touch. The doctor said it had been ulcerated but the ulcer had disappeared. There were some mucous patches over the velum. There was considerable swelling of the anterior cervical glands, noticeably worse on the right side. There was some eruption over the palms and she said she had a slight eruption over her body. The doctor was quite stunned when I called his attention to these stigmata of syphilis and reproached himself for not having sooner made a diagnosis so as to have sooner instituted proper treatment.

The husband of the patient was a traveling salesman and had had a sore mouth and some ulcers on his lips when home a short time before and it was from this source that we thought the contagion had been conveyed to the mouth of the patient. The reputation of the woman was, and is, of the very best and on that account the doctor had no thought of syphilis in connection with her illness.

Case 2. Miss Blank, aged twenty-three, was brought to me by her family physician for an intractable "sore throat" of some weeks' duration. She had a "cold sore" on her lower lip which had resisted all usual remedies. There was not much pain in the throat but a persitent ear ache seemed to be caused by the throat trouble. On the inferior labium, to the left of the center, was a swelling

indurated to the touch and ulcerated at the apex. On the left tonsil was a grayish ulcer one-third of an inch in diameter slightly elevated and surrounded by a zone of indurated tissue. Each of these lesions had been present more than a month and had somewhat diminished in size. There had never been any pronounced pain except in the left ear which had evidently radiated from the left tonsil. The cervical glands at the angle of the jaw were swollen and tender to pressure. My diagnosis of hard chancre of lips and tonsil was received with doubt by her physician but the appearance of a macular syphilide over the abdomen convinced both patient and doctor of the character of the trouble. Then we hunted for the cause and finally traced it to kissing a man "who had been sick and was on his way to Hot Springs, Arkansas, for treatment." The character of the girl we all considered above reproach. She was put on constitutional treatment and her disease fully explained to her. After about three years she was married and is now an apparently happy and healthy mother and her children show no trace of the disease.

Case 3. Miss X., aged nineteen, was sent to me for stenosis of one naris. Examination revealed an obstructing septal shelf. But on the posterior pharyngeal wall was a long gray ulcer projecting up into the naso-pharynx. I asked her doctor the cause, but he had not seen it before and he had supposed all her trouble was due to the intra nasal obstruction. There was a distinct enlargement of the cervical glands and she gave a history of having had a slight eruption over the body some time before. The family doctor was badly puzzled as he knew the girl well and believed her to be virtuous. So he immediately jumped to the conclusion that it was a late heredity development and so told her father. On her next visit, she lived 100 miles from Cincinnati, I examined her more carefully as I did not believe in the doctor's late hereditary theory. I then found a scar on her lip which she said had resulted from an intractable sore which had been present about a year before and had lasted more than a month. The family doctor then remembered the sore lip and had no hesitancy in calling it the initial lesion acquired from kissing a syphilitic sweetheart.

Case 4. Miss S., aged sixteen, consulted me for a persistent sore on the inferior labium. It had been present four weeks and despite the heroic(?) efforts of her family doctor, a homeopathic practitioner, it was not improving. There was present a large

swelling to the right of the medium line. The center was ulcerated and the case was indurated. The anterior cervical glands on the right side were swollen and tender. After much talk I convinced the doctor that the lesion was a hard chancre, and proper treatment was instituted. About a month after another doctor called on me and said he knew of this case and he could furnish me with the missing link. A short time before the girl had the lip sore he was treating a young man, who was regularly calling on her, for secondary syphilis and he had persistent mucous patches in mouth and on lips. The most reasonable theory then was that by kissing the girl he had inoculated her with syphilis.

From these reports it will be observed that the diagnosis was not readily made. Pain was not a prominent symptom, as in the cases reported by Rhodes (*Am. Lar. Journ.*, 1901.) "The sooner the profession recognize that syphilis is not merely a venereal disease, the better for all."

That these cases are frequently overlooked goes without saying when we consider how frequently the reported cases are revealed by accident.

Dr. O. M. Smith, (*Journ, Cutaneous Diseases*, 1906,) before the Boston Dermatological Society reports a case of roseola over the trunk while on the tonsil was a deep ulcer surrounded by a zone of redness and infiltration. In the discussion that followed all the members present agreed that "in this part of the body a diagnosis was always difficult to make."

In the discussion of Dr. Rhodes' paper before the American Laryngological Association Drs. Bryan, Farlow and Leland all agreed as to the difficulty in diagnosis. Of course one must remember that most of these cases are first seen by the family doctor who is not an expert in differental diagnosis of such diseases.

Pain in the tonsil was not complained of in my two cases.

Dr. Shields reported a case at the Cincinnati Academy of Medicine (*Cin. Lancet Clinic*, 1904,) where there was but little pain. But in a case reported to the Academy the same year by Dr. R. C. Jones there was considerable pain which was also present in Dr. Max Thorner's case reported to the same society in 1897. In Dr. Rhodes' cases pain was a prominent factor. From these diverse reports it is easy to conclude that pain may or may not be present in chancres of the tonsil.

The enlargement of the cervical glands is always present and entirely out of proportion to the amount of affection of the tonsil. This adenopathy, together with induration of the tissue surrounding and underlying the ulcer is almost pathognomonic, and should at least always cause us to interrogate the entire body for other symptoms. In case of doubt the secondary symptoms will appear long before the disappearance of the primary lesion.

Perhaps of the greatest interest to us in these cases is their etiology. My cases were almost certainly due to kissing, as were two of Dr. Rhodes' cases. Bestial practice was given by Dr. Thorner as the cause of his case. Bruhns (*Berl. Klin. Wchnschr.*, 1900,) says that only rarely is hard chancre in the mouth acquired by bestial practices. The usual method of contagion was by kissing a person who had syphilitic lesions on the lips or tongue, or by drinking vessels or utensils used in eating. Dr. Richards thinks that the contagion is most frequently conveyed to the tonsils by a pipe or drinking vessel. Dr. Bulkley says that the virus of syphilis is one of the most energetic of animal contagions. All unite in giving out the warning that syphilitic ulcers of the mouth are exceedingly virulent and are frequent means of conveying the poison to innocent parties.

PAPER:

A STUDY OF THE ETIOLOGY OF INFLAMMATORY DISEASES OF THE NOSE AND ACCESSORY SINUSES.

WILLIAM LINCOLN BALLINGER, M. D.

Introductory Remarks.—I have long been of the opinion that the causes of the inflammatory diseases of the nose and accessory sinuses have not been systematically presented in any work on rhinology. Believing myself incapable of elucidating the subject in a logical and convincing manner I have, heretofore, refrained from formulating my ideas in writing. In my lectures at the College of Physicians and Surgeons, I have more than once given utterance to them, and have finally concluded to place them in writing with a view of inviting my confreres to criticise, or to re-

fute them if they do not appeal to their judgment or coincide with their experience.

My views are based upon clinical observations and upon well known laws relating to inflammatory processes. I am emboldened, therefore, to present them to this body of distinguished rhinologists for their approval or disapproval.

INFLAMMATION.

Before discussing the causes of inflammation, it will be well to define what inflammation is.

Acute Inflammation.—Acute inflammation is a three-fold reaction excited by the presence of certain noxa, or irritant material, in the tissues. The noxa or irritant is usually a pathogenic microorganism and its toxin, or it may be of chemical or traumatic origin. When of chemical or traumatic origin the irritant primarily consists of the dead or broken down cells of the tissues.

Dead or broken down cells when present in the tisues in excess become foreign bodies, and as such, a reaction of the living cells is excited for the purpose of eliminating them from the body. Furthermore, the dead cells in the process of disintegration give off a ferment or chemical substance which also excites a reaction, the purpose of which is to free the tissues of its presence. The reaction thus far excited is directly traceable to the presence of dead and disintegrating tissue cells. Ordinarily, after a short time, a secondary irritant gains entrance to the injured tissues and becomes the more important factor in the reactionary process. That is, pathogenic bacteria infect the impaired tissues so that in nearly every acute inflammatory process, whether it is due to primary infection or to chemical or mechanical trauma, pathogenic microorganisms must be regarded as the paramount exciting or noxious agent causing the reaction of inflammation.

The reaction of inflammation is, therefore, an increased physiological activity of the living tissues of the body for the purpose of disposing of a noxious or irritant substance or organisms that have invaded them in excess of the normal quantities.

The reaction of acute inflammation is a three-fold process, namely:

1. Increased hyperaemia.
2. Increased nutrition (increased resistance.)
3. Increased leukocytosis.

1. Increased hyperaemia is a constant and important reaction as through it the cells are provided with the extra nutrition they need under conditions of stress. The increased blood supply also stimulates and facilitates the increased migration of leukocytes, and it flushes the poisoned area and dilutes the noxious substance and thus reduces the intensity of the irritation. The hyperaemia is nearly always passive in type. It has been demonstrated that passive hyperaemia is more potent in overcoming bacterial irritation than active hyperaemia, though active hyperaemia when well established is also very efficient.

2. Increased nutrition of the tissues is prompted by the hyperaemia for obvious reasons. They are under stress because of the presence of noxious substance, and need extra nutritional facilities. Their vital force, or resistance, is not equal to the emergency placed upon them and upon their resistance depends the issue of the warfare. Their means of defense may be characterized as two-fold, namely, (a) their ability to envelope and digest micro-organisms, and (b) their ability to produce and emit a biochemical substance or ferment, the purpose of which is to weaken or destroy their foe. This all requires increased nutrition (blood) which begets increased powers of resistance.

If the nutrition is not adequate for these purposes the micro-organisms and their toxin or biochemical irritant, may cause destructive, and what we are accustomed to call pathogenic changes in the tissues.

3. Increased leukocytosis is also an important reaction of inflammation. While the function and modes of activity of the leukocytes is not fully understood it has been fairly well demonstrated that the polymorpho-nuclear leukocytes envelope and destroy bacteria, while the lymphocytes envelope and destroy broken down cells. Other cells as the fibroblasts also participate in these functions under certain conditions. I have not the time to enter into a discussion of all the processes included in the reaction of inflammation and only desire to briefly suggest the more important and well known processes in order to prepare our minds for a clearer understanding of the etiology of the inflammatory diseases of the nose and accessory sinuses.

Parenthetically I wish to add one additional statement concerning the adequacy of the reaction of inflammation. According to Adami the reaction of inflammation may be of three types:

1. Adequate reaction.
2. Inadequate reaction.
3. Excessive reaction.

The reaction is usually inadequate. That is, the increased hyperaemia, cell nutrition and migration of leukocytes is insufficient to dispose of the pathogenic micro-organisms before they have caused considerable damage to the tissues. It follows, therefore, that in the treatment of inflammatory diseases, the reaction of inflammation should be promoted rather than diminished. By so aiding the defensive and offensive activities of the tissues, the bacteria, their toxins, and the broken down tissue cells may be speedily removed, and a cure effected.

Inflammation Affecting Mucous Surfaces.—Adami says: "The main distinguishing feature of the mucous surface is the presence there of a layer of mucous cells, cells of a glandular type, capable when stimulated of forming and discharging relatively large amounts of mucin. The hyperaemia, the exudation of serum, the migration of leukocytes, all these occur in the submucous layer just as in the subserous layers. The changes in the reaction are due solely to the interposition of this layer of mucous cells. There is, in the first place, a more definite basement substance interposing a certain amount of resistance to surface exudation. The layer of mucous cells is more complicated, and although the fully developed cells may be discharged, they are apt to remain relatively undifferentiated 'mother cells' at the base; or otherwise the same intensity of irritation does not lead to as extensive a denudation. And, thirdly, by the combined action, it may be, of the irritant and of the hyperaemia, the fully formed mucous cells are stimulated to produce increased amounts of mucin, so that an inflammation of moderate grade is characterized by an abundant amount of mucinous discharge rather than of fibrinous deposit."

"We speak of such a moderate case, with exudation of serum containing abundant mucin, cast-off mucous cells, and relatively few leukocytes, as a 'catarrhal inflammation,' if there be sufficient leukocytes extruded, the character is altered to that of a 'mucopurulent inflammation;' if more severe, with complete destruction of the mucous membrane proper, then, as in serous surfaces, there is the same tendency for the leukocytic exudation to favor a deposit of fibrin upon the surface, and then we obtain a 'membranous inflammation.'"

"Despite the fact familiar to all that diphtheria is a disease set up by a specific bacillus, and the equally well-known fact that a like membranous inflammation may be induced by several forms of microbes, we still commonly speak of such a membrane as being diphtheritic. It would be better to confire this term purely to cases in which we know that the bacillus diphtheria is the causative factor; failing this, we may accept the term diphtheritic as covering all such membranous inflammation, and employ the term diphtherial for such cases as are of pure diphtherial origin.

"Further, if there be yet more severe destruction of the surface cells, this may go on to ulceration. Where we have pyogenic organisms present, there is a dissolution and breaking down of any fibrin that is formed and consequent absence of a membrane. In such cases there is a distinct tendency for the process to extend in the submucosa beneath the still intact mucous membrane, the part becoming infiltrated with pus. This form is spoken of as 'phlegmonous inflammation.'"

Chronic Inflammation.—The reaction of chronic inflammation consists of the following phenomena:

a. Slightly increased hyperaemia.
b. Slightly increased cell nutrition.
c. Slightly increased migration of leukocytes.

It is needless to add that the reaction is inadequate to remove the noxa or irritant which according to pathologists is usually bacteria of low virulence. There is usually a local obstructive lesion of the nose in chronic inflammation.

A product of chronic inflammation that is always present is the proliferation of fixed cells, usually of the least differentiated type, namely, connective tissue cells.

THE ETIOLOGY OF INFLAMMATORY DISEASES OF THE NOSE AND ACCESSORY SINUSES.

Having thus briefly defined inflammation we are prepared to discuss its causes.

The causes of inflammatory diseases of the nose and accessory sinuses are divided into two groups, namely:

1. Exciting causes.
2. Predisposing causes.

1. *Exciting Causes.*—The exciting causes are bacteria, and chemical and traumatic destruction of tissue cells. This phase of the subject has already been discussed under Inflammation and will not be

dwelt upon in this connection further than to say, that, pathogenic bacteria can not irritate the tissues of the body so long as the resistance of the cells is normal, that is, so long as they are healthy. There may be an exception to this rule when the germs are exceptionally virulent, though this is rare. Virulent pathogenic bacteria are constantly present in the upper respiratory tract, though they are harmless until the resistance of the cells is lowered by some intracorporial or extraneous influence.

2. *Predisposing Causes.*—There are many predisposing causes of inflammatory diseases of the nose, some of which are best explained by grouping them around a well recognized physiopathologic law, namely, *When the drainage and aeration of a mucous membrane lined cavity is impaired or blocked, the conditions are favorable for the growth of pathogenic bacteria.*

If this is true, each case of inflammatory disease of the nose and accessory sinuses should be examined to ascertain if the drainage and aeration of these spaces are impaired or blocked. If they are, the obvious therapeutic duty is to remove the obstruction by such remedial measures as will best accomplish the purpose. These measures may be either medicinal, hygienic or surgical.

If, on the contrary, no obstructive lesion is found, other causes for the lowered resistance of the tissue should be sought for. If the inflammation is a primary acute one, and the lowered resistance is due to shock from exposure, it may be useless to attempt to remove the cause as it was transient. The immediate duty in such a case is to promote the reaction of inflammation and thus check the inflammatory process. As Adami so aptly says, the way to cure inflammation is to increase it.

In order to logically approach the consideration of the causes of the lowered resistance of the mucous membrane of the nose and accessory sinuses they should be divided into two groupes, namely:

a. Extranasal. b. Intranasal.

EXTRANASAL PREDISPOSING CAUSES.

Age seems to exert some influence upon the resistance of the nasal mucous membrane. Young children and young adults are more frequently subject to inflammatory diseases of the nose and accessory sinuses than those of more advanced years. This is, no doubt, due in part to indiscretion, as the improper care and protection of the body from the inclemencies of the weather. Persons of

more mature years have more mature minds and better judgment, and they do not expose themselves needlessly, as in youth and childhood. Then, too, the tissues acquire a resistance, or immunity to the noxious irritations.

Sex, perhaps, exerts some influence on the occurrence of inflammatory processes. Males are more exposed and more reckless than females, hence they are more often affected by inflammatory diseases. They are more pugilistic and more often have broken noses and consequent nasal obstruction than females.

Climate undoubtedly influences the occurrence of inflammatory processes. In regions where there is much cold wet weather with sudden changes of temperature, and of hygroscopic conditions of the atmosphere, it is more difficult to protect the body, particularly the feet, from the shock incident to such exposures. The shock thus sustained by the vasomotor nervous system leads to a lowered resistance of the mucous membranes, especially of the nose and accessory sinuses, hence the growth of bacteria in these regions is favored.

Exposure, especially unusual or unequal exposure of the body to damp cold or other atmospheric and metallurgic conditions, weakens the resistance of the tissues. The exposure of the feet to damp and cold is a most fruitful source of rhinitis and inflammations elsewhere in the body. Draughts striking a single portion of the body are detrimental to the resistance of the tissues much more than when the whole body is thus exposed. Within certain limitations the exposure of the whole body often has a tonic effect, as all the animal mechanisms of the body are equally and simultaneously stimulated. When partial exposure is experienced only a portion of the mechanism is stimulated and an imbalance of the functional activities results. That is, there is confusion and havoc in the cellular activities, the nasal expression of which is often some form of inflammation.

The *clothing* is an important factor in maintaining or lowering the resistance of the mucous membrane of the upper respiratory tract. Too much is as productive of evil as too little clothing. If too much is worn the skin is rendered sensitive to slight exposure, and if too little is worn the body is subjected to continual stress, and exhaustion of the vital forces results. Either condition prepares the soil for the growth of pathogenic bacteria in the respiratory passages. Perhaps the most vulnerable part of the body is the

feet, through the soles of which course large blood vessels. Anyway, cold, wet feet are a common cause of acute rhinitis and sinusitis.

The proper selection of *underwear* is a much mooted question. Wool is advocated by some, while linen or linen mesh is strenuously recommended by others. In the meantime most persons buy cotton for summer and cotton and wool mixtures for winter wear; not because they believe they are the best, but because they are cheaper.

My ideas on the subject are as follows: Linen absorbs moisture better than either cotton or wool, and is, therefore, better for summer wear. Wool is warmer than either cotton or cotton and wool mixtures and is better for winter wear. Some persons perspire easily in winter and for them linen should be worn next to the skin. If this does not retain enough body heat light wool should be worn over the linen underwear. Cotton or cotton and wool mixtures are perhaps never preferable to wool and to linen and wool combinations for the winter months.

The outer garments should be medium weight for the winter months, the over garments being depended upon for extra protection for outdoor wear. If the indoor clothing is too heavy the skin becomes tender and subjects the wearer to shock upon undue exposure when out of doors. The underclothing and outer garments should, therefore, be selected for their absorptive and heat-retaining properties. Hard and fast rules cannot be laid down in reference to the clothing, each subject being a law unto himself. The aim should be to so regulate the clothing as to avoid either extreme, as to do otherwise subjects the system to shock, and thus lowers the cellular resistance and prepares the soil for the growth of micro-organisms and inflammation.

The *digestive tract* is by some writers justly held responsible for inflammatory processes of the upper respiratory tract. If the processes of digestion and nutrition are imperfectly performed, noxious material enters the vascular and lymphatic circulation and thus places extra stress upon all the fixed and migrating cells of the body. Lowered resistance therefore naturally follows.

Certain *constitutional diseases* likewise produce a lowered resistance of the tissues, including the mucous membrane of the nose and accessory sinuses. Diabetes, syphilis and all diseases due to faulty metabolism especially affect the tissues of the respiratory tract and predisposes them to infection and inflammation.

Heredity probably has no direct influence in the predisposition to infectious and inflammatory diseases of the nose. Indirectly it may have such an influence. That is, certain anatomical conformations of the nasal chambers may be transmitted from parents to the child and thus lead to a predisposition to infection and inflammation.

Adenoids may interfere with the drainage and aeration of the nose and accessory sinuses, or inflammation focalized in them may lower the resistance of the mucous membrance and the nasal and accessory sinuses and thus predispose to infection and inflammation. These and other extranasal influences may prepare the soil for the growth of pathogenic bacteria in the nose and accessory sinuses and may eventuate in empyaema of the sinuses without obstructive lesions in the nose. Whatever the case of the lowered resistance of the mucous membrane the result is the same.

I do not wish to be understood as saying that infection and inflammation always follow a lowered resistance of the nasal mucous membrane. I only claim that a lowered resistance predisposes to such a process. The virulence of the micro-organisms present and perhaps other conditions enter into the equation.

INTRANASAL PREDISPOSING CAUSES.

In this connection I wish to repeat the physio-pathological law which largely explains the occurrence of infection and inflammation of the nose and accessory nasal sinuses, namely,

Cavities lined with mucous membrane are predisposed to inflammation when their drainage and aeration are obstructed.

From experience we know that when such obstructions are present and are removed either by local application or by surgical interference, relief often promptly follows.

Let us direct our attention, therefore, to some of the obstructive lesions of the nose which predispose the mucous membrane to infection and inflammation.

Obstruction of the Lower Portion of the Nose.—I desire first to call attention to a fact that has long impressed me as very important, namely, that obstructions in the lower portion of the nasal cavity have a different clinical significance from obstructions located higher in the nasal passages. I also wish to call attention to the clinical significance of anterior obstructions as contrasted with obstructions otherwise located.

Obstruction of the Inferior Portion of the Nose.—Obstruction of the inferior portion of the nasal passages causes an approximation or an impingement of the inferior turbinal against the septum, at least at certain points. The pressure may be either intermittent or constant. The question of greatest importance is, How does such an obstruction affect the drainage and aeration of the nose and sinuses? As most of the mucous membrane of the nose and sinuses is located above the inferior turbinal, it is obvious that aeration is but little affected by such an obstruction. The pathway of the inspiratory current is largely limited to the middle and superior meatuses of the nose, and, inasmuch as an obstruction located inferiorly does not materially occlude the inspiratory tract, there is comparatively little disturbance of function. Furthermore, the drainage of the secretions is not materially blocked. The usual obstructive lesion in this region is a spur or ridge on the septum. The ridge is rarely equally prominent along its entire length. On the contrary it presents one or two prominent spines or knuckles which approximate or impinge against the inferior turbinated body, thus leaving wide gaps through which the secretions may drain to the floor of the nose without marked impediment.

The practical deduction to be drawn from these facts is, that an obstruction in the lower portion of the nose does not markedly reduce the resistance of the mucous membrane, especially in the upper portion of the nasal chambers and in the accessory sinuses. It does, however, have some influence in this direction, and in a degree predisposes to infection and inflammation. The crests of the spines or knuckles may accumulate secretions, which become dessicated in the form of moist or dry crusts. The tissue cells beneath the crusts are injured, and their resistance lowered, and to this extent there is a predisposition to infection and inflammation. Furthermore, the impingement of the spur or spine against the outer wall of the nose causes traumatic injury and results in some infection and inflammation. The irritation is not usually pronounced and only causes an increased hyperaemia and nutrition of the tissues.

Obstructive lesions in the lower portion of the nose, therefore, may cause a turgescence of the mucous membrane, which is richly supplied with erectile tissue (the swell bodies), which, after a more or less prolonged period, may result in hypertrophy. In the early or turgescent stage the condition is called turgescent rhinitis; in

the latter stage, it is called hypertrophic rhinitis. If, however, repeated infection occurs, the irritation is of a different type and causes hyperplastic changes.

Unfortunately, however, a deviation of the lower portion of the septum is usually accompanied by a deviation of the upper portion of nose in the region of the middle turbinal. When this is the case the type of inflammation is radically different from that present in an uncomplicated lower deviation. That is, a deviation in the region of the middle turbinal often obstructs the drainage and aeration of the superior meatus, and of all, or nearly all, of the nasal accessory sinuses. The secretions are retained, undergo decomposition, liberate a ferment and irritate the mucous membrane. In brief, the inflammation is attended by the proliferation of the least differentiated of the fixed cells of connective tissue cells. In other words, hyperplasia of the mucous membrane occurs. This is known as hyperplastic rhinitis. The irritation from the middle turbinal region may extend by continuity of tissue to the inferior turbinal and cause hyperplasia of this structure as well. Hence, hyperplastic rhinitis often involves both turbinated bodies. In simple deviations, however, limited to the lower portion of the nasal chambers, the inflammation is usually of the hypertrophic type.

Obstruction of the Anterior Portion of the Nose.—Deviation of the anterior portion of the septum from traumatism is a common cause of obstruction of the anterior portion of the nasal chamber. The relationship it bears to inflammatory processes of the nose and accessory sinuses is interesting and instructive. An anterior deviation does not interfere with the drainage of the secretions except in so far as it may affect the mechanical force of the respiratory currents of air. The mechanical force of the inspired air is especially manifested in the region of the infundibulum and posterior ethmoidal cells where the inspiratory current sweeps over the hiatus semilunaris and the ostia of the posterior ethmoidal cells and causes slight rarefaction of the air within the sinuses drained by these openings. The mechanical impact facilitates the flow of secretions from the ostia and hiatus semilunaris and thus prevents dessication and blockage of these openings. To this extent obstructive anterior deviations of the septum interfere with drainage.

The aeration upon the obstructed side is, however, very materially affected. The slight interference with the flow of the secre-

tions caused by the absence of the mechanical impact of air results in a moderate retention of secretions. Decomposition of the secretions may therefore take place and cause a lowered resistance of the mucous membrane and thus establish a predisposition to infection and inflammation.

When the ridge or spur in the lower portion of the nose extends well forward into the vestibule, it also interferes with the aeration drainage as described in the preceding paragraph.

When either type of anterior obstructive deviation is present, another and more important etiological factor must be taken into consideration, namely, the rarefaction of air posterior to the obstruction. Air being unable to enter the nostrils rapidly enough during the descent of the diaphragm is rarefied, or a state of negative air pressure is established. This, according to Bier's theory, should prevent serious inflammatory processes, as the negative air pressure thus produced promotes the reaction of inflammation and thus prevents serious inflammatory disease. Doubtless the negative pressure thus automatically produced does exert a favorable influence upon the inflammatory process excited by the lack of aeration and the slight retention of the secretions. Thus, strange as it may seem, the anterior obstructive lesion predisposes to infection and inflammation and at the same time tends to cure it.

Clinically, I have often noted the comparatively slight inflammatory disease of the nasal mucous membrane present in simple anterior deviations.

The chief departure from the normal is a turgescence or a hypertrophy of the inferior turbinal. Little pathologic change is present in the middle turbinal region unless there is an associated obstruction in that location. The negative air pressure easily accounts for the turgescence of the erectile tissue of the inferior turbinals. After a prolonged duration of the turgescence, whether intermittent, alternating or constant, hypertrophy occurs as a result of the increased nutrition.

Obstruction of the Middle Turbinal Region.—Obstruction in this portion of the nasal chambers is productive of more serious inflammatory disease of the nose and accessory sinuses than obstruction in any other portion of the nose. This is apparent when we recall the fact that the ostia of the posterior ethmoidal and sphenoidal sinuses open into the superior meatus above the middle

turbinal, while the frontal, anterior ethmoidal and maxillary sinuses drain into the middle meatus beneath the middle turbinal.

If the septum is deviated so as to press against, or approximate near to, the middle turbinal, the olfactory fissure is blocked and the drainage of the posterior ethmoidal and possibly of the sphenoidal cells is interfered with.

Clinically I have noted the presence of two types of deviations of the septum that close, or nearly close, the olfactory fissure. One is a bowing of the perpendicular plate of the ethmoid bone and triangular cartilage, and the other is a thickening of the septum in the region of the middle turbinated body. The bowed septum is thin and easily corrected by the submucous resection of the septum, whereas the thickened septum often involves only the mucous membrane and is more difficult to correct.

In some subjects there are large pneumatic spaces in the middle turbinal which may either close a part or all of the olfactory fissure, or they may encroach upon the hiatus semilunaris beneath it. In the first instance the drainage and aeration of the superior meatus of the nose, and in the second instance the drainage and aeration of the frontal, anterior ethmoidal and maxillary sinuses is impaired.

A large bulla ethmoidalis projecting medianward and downward may obstruct the hiatus semilunaris, and thus obstruct the drainage and aeration of the cells draining into the infundibulum, namely, the frontal, anterior ethmoidal and maxillary sinuses.

Likewise the occasional pressure of cells in the inner wall of the infundibulum or uncinate process of the ethmoidal bone may block the infundibulum and cause serious inflammatory disease of the frontal and anterior ethmoidal cells and the maxillary antrum.

In about 50 per cent. of cases the fronto-nasal canal does not communicate with the infundibulum, but opens directly into the middle meatus more anteriorly. In these subjects an enlarged projecting bulla ethmoidalis and cells in the uncinate process would not block the drainage and aeration of the cells draining through the fronto-nasal canal, namely, the frontal and anterior ethmoidal cells. The ostium of the antrum, however, may be obstructed, as it usually opens into the infundibulum.

THE RESULTS OF HIGH OBSTRUCTIONS.

In the Nose.—When the olfactory fissure is obstructed by either septal or turbinal deformity, the drainage of the secretions and the

aeration of the posterior ethmoidal and sphenoidal sinuses is impaired. The secretions are retained and undergo retrograde changes. The mucous membrane is bathed in the secretions and becomes impaired and its functional activity and its resistance are lowered. The biochemical substances liberated in the process of decomposition constantly irritate the mucous membrane, especially of the middle turbinated body. Acute infection occasionally occurs. During the intervals between the acute inflammatory processes a mild staphylococcal or other infectious inflammation persists. Under these conditions there is a proliferation of fixed cells in the tissues, usually the least differentiated of the fixed cells, viz.: connective tissue cells.

The results of these influences in the nose is known as Hyperplastic Rhinitis, which chiefly involves the middle turbinated body, though it often extends to the inferior turbinal as well.

In the Posterior Ethmoidal and Sphenoidal Cells.—The partial or complete closure of the olfactory fissure, and the consequent retention of the secretions of the superior meatus, and the ethmoidal and sphenoidal sinuses draining into it, cause hyperplastic changes in the mucous membrane, not alone of the middle turbinal, but of the superior meatus and of the accessory sinuses opening into it. The conditions thus produced are favorable for infection and inflammation. The inflammatory process may be either catarrhal, purulent, fibrinous, phlegmonous or suppurative in type, and in each instance it is in part due to pathogenic micro-organisms.

The sinusitis thus excited may continue for years without engaging the attention of either the patient or physician. Headache and slight dizziness, aggravated upon stooping, may be the only symptoms complained of, except, possibly, recurrent attacks of acute coryza. Or the sinusitis may be distinctly and frankly purulent with copious discharge into the epipharynx, and possibly to some extent through the olfactory fissure.

Atrophic rhinitis with ozena is, in my opinion, in adults, often a suppurative sinusitis with atrophy of the mucous membrane. Space does not permit of a full discussion of this phase of the subject. Personally I have repeatedly overcome the ozenic secretion by treating the case as though it were a suppurative sinusitis. I have made skiagraphs of several cases of atrophic rhinitis with ozena, and without exception they have shown the existence of sinus disease. This does not, of course, determine which was primary, the

atrophic rhinitis of the sinusitis. My opinion is largely based upon the results following the treatment for the sinusitis.

Obstruction Due to the Bulla Ethmoidalis, Cells in the Middle Turbinal and Uncinate Process.—As previously stated, a large bulla ethmoidalis may occlude the infundibulum and thus block the drainage and aeration of the maxillary, frontal and anterior ethmoidal cells. This, as heretofore explained, causes the retention of the secretions and lowered resistance of the tissue, thus establishing a predisposition to infection and inflammation.

Cells in the middle turbinated body and uncinate process likewise may block the infundibulum and cause similar results. The exceptions have been referred to wherein the fronto-nasal canal opens directly into the middle meatus anterior to the infundibulum.

It appears, therefore, that there are several factors entering into the causation of inflammatory diseases of the nose and accessory sinuses. The exciting cause is nearly always pathogenic microorganisms, while the predisposing causes are numerous extranasal influences which are often combined with obstructive lesions in the nose. The latter should always be studied with reference to whether they interfere with the drainage and aeration of the nose and accessory sinuses. If only extranasal causes of lowered resistance are found, the treatment should be addressed to their removal; and if, in addition to the extranasal influences, obstructive lesions are found, they should be corrected by probing or by surgical interference.

CONCLUSIONS.

1. Acute inflammation is usually a threefold reaction excited by pathogenic bacteria and their toxins, namely:

(a) Increased passive hyperemia.
(b) Increased nutrition of the tissues.
(c) Increased migration of leukocytes.

The reaction of acute inflammation is the response of Nature's forces for the purpose of destroying the bacteria and their toxins.

2. The reaction of inflammation is usually inadequate to quickly remove the infective bacteria and their toxins, hence the inflammation is continued for several days, or it may be indefinietly prolonged.

3. Chronic inflammation consists of the same reactions in much less degree, and is still further characterized by the proliferation of fixed cells into the tissues, notably connective tissue cells.

4. The exciting cause of inflammation is generally some pathogenic micro-organism.

5. Pathogenic bacteria do not *per se* cause inflammation. There must be a lowered resistance of the tissues before they will rapidly multiply and produce inflammation.

6. Anything that lowers the vitality or resistance of the mucous membrane of the nose and accessory sinuses predisposes it to infection and inflammation.

7. The extranasal influences that lower the vitality of the mucous membrane are: Sex, climate, exposure, improper clothing, digestive disorders, constitutional diseases and dyscrasia, hereditary anatomical peculiarities of the framework of the nose, adenoids, etc., etc.

8. The intranasal predisposing causes of inflammation of the mucous membrane of the nose and accessory sinuses are, perhaps, best explained by the well-recognized law that *Obstruction of the drainage and aeration of mucous-lined cavities predisposes them to infection and inflammation.*

The character of the inflammation and the end result is partially determined by the location of the obstruction in reference to the various tissues of the nose and to the accessory sinuses.

9. Anterior and inferior obstructions more often cause turgescent and hypertrophic rhinitis, as they do not materially interfere with the drainage of the secretions and therefore cause very little or no irritation.

10. Obstruction higher in the nose, in the region of the middle turbinal and the infundibulum causes the retention of the secretions and interferes with the aeration of the superior meatus and the accessory sinuses, thus lowering the resistance of the tissues, and establishing a marked predisposition to infection and inflammation of the nasal and accessory sinuses. The inflammation may be catarrhal or suppurative and acute or chronic in type.

11. The long-continued mild irritation excited by obstructive lesions in the middle turbinal region often results in hyperplastic rhinitis, which may be limited to the middle turbinal, though it may extend to the inferior turbinal.

12. Inflammation also extends to adjacent parts by the continuity of tissue, hence it may extend from one part of the nasal mucous membrane to another, or it may extend from the nasal mucous membrane to the sinuses.

MEETING OF WESTERN SECTION, HELD IN DENVER, COLO., FEBRUARY 16, 1907.

EXHIBITION OF CASES:

1. In the absence of Dr. Levy, Dr. Bane exhibited Dr. Levy's patient. Mrs. C., aged 28, with posterior hypertrophy of the inferior turbinals, the right being larger than the left; the hypertrophied portion of the turbinals hung down loosely from the posterior nares, were dark purplish in color and very dense; the right side was removed, the operation requiring about half an hour and being attended with great difficulty on account of the dense nature of the tissue. A microscopic examination of the tumor showed an excess of fibrous tissue; the blood spaces less abundant than normal; scarcely any mucous glands; the connective tissue cells in the centre of the tissue, adult in character, but less so toward the periphery. At the periphery were many true small round cells with a few polymorphonuclear leukocytes; The surface epithelium was fairly normal.

2. Dr. Bane exhibited an ear patient, a man aged 29 years. History of double otitis media from concussion in 1898. The ears were treated but not cured. On July 26, '05, severe pain developed in the right ear and side of the head, accompanied with dizziness. Hearing with the right ear failed entirely on that day. When seen Aug. 22, 1905, he was having severe pain in and about the right ear. Hearing was nil. A small perforation existed in the upper posterior quadrant of the drumhead. The enlargement of the perforation with a probe permitted of a free escape of pus into the canal. The radical operation was advised and performed the same day. Foul pus flowed from the antrum as soon as it was entered. The bone was eburnated. The Panse flaps were stitched backward. The wound was closed and the cavity packed through the canal. The patient suffered with severe pain in the head and had attacks of dizziness following the operation. The ear healed slowly, not ceasing to discharge for five weeks. A relapse occurred a year after the operation, in that pus developed in the upper

anterior portion of the middle ear. Evacuation was followed by a permanent opening in a membranous formation that is readily seen in the ear. The ear is perfectly dry. Attacks of dizziness with staggering gait are not infrequent. He is liable to fall if he attempts to carry a load on his shoulder. The left ear is apparently diseased in the attic, though the drumhead is intact. Hearing is nil for the voice in the left ear. General health is excellent at the present time.

3. Dr. Bane exhibited a patient upon whom he had done a double mastoid in the past six weeks. A robust man of 44 years. Inflammation had developed in the left ear the third week in December, '06. The ear discharged for about ten days, when it diminished. Then some swelling over the upper part of the mastoid appeared. There was but moderate tenderness to pressure. The temperature rose to 100 degrees F. on January 2nd, 1907. The canal was so narrowed by sagging of the canal wall that the drumhead could not be seen. The patient was admitted to the County Hospital on Jan. 3d, and operated Jan. 4th. A large subperiosteal abscess was encountered; also two perforations in the cortex. One led into the antrum and the other to a large abscess over the lateral sinus. When exposed, the sinus was found to be covered with granulation tissue. Most of the mastoid was diseased. The cavity was lightly packed with iodoform gauze. On Jan. 21, '07, the patient stated that he had an occasional pain in and above the right ear. Examination elicited some tenderness back and above the auricle. The canal wall was sagging so that the drumhead could not be seen. The transillumination was poor. There was no history of acute inflammation of the ear and there had not been any discharge. Operation on Jan. 27th, '07. When the periosteum was pushed forward, exposing the bony canal, some pus escaped from between the membranous and bony canal. The outer table was dense and dry for three-eighths of an inch, when a diseased area was entered. A large cell was found over the middle ear, that contained pus, and a perforation existed in the floor of the cell communicating with the middle ear. The antrum was diseased and necrotic, the disease extended from the antrum to the tip, which was covered throughout with dense bone. The points of special interest in the left ear were the advanced stage of the disease without any discharge from the ear, and so little complaint having been made of pain.

4. Dr. Bane presented Mrs. J. W. S., aged 51, who came Jan. 8, 1907, complaining of having had disease of the ears for the past 20 years. Discharge from both ears at intervals during that period. The left ear had pained her constantly for three weeks, but had not discharged. Hearing 2-60ths. The drumhead was intact. There was tenderness of the left mastoid and light was not transmitted through it, but was transmitted through the right. There was free muco-purulent discharge from both nostrils. The left antrum of Highmore was tender and decidedly dull to transillumination. Pulse 90, and temperature 99 degrees F. On January 16th, '07, she reported having had a constant tingling sensation over the body for the previous 12 days. No pain in the left ear for three days. She was advised to have the left maxillary sinus emptied and the left mastoid explored. Patient is present today for examination. The examination revealed almost no dullness of the sinus, and yet no operation had been done.

PAPER:

THE ANTRUM OF HIGHMORE AS AN ETIOLOGICAL FACTOR IN THE PRODUCTION OF HAY-FEVER, (SO CALLED.)

JACOB E. SCHADLE, M. D.

THE PREMISES.

The following paper is presented for the purpose of drawing special attention to a certain phase of a familiarly known disease, the cause and treatment of which have always been perplexing to understand. According to recent experiments, the disease appears to be based on anatomic deviations in its pathologic aspects. These anatomic malformations act both as predisposing and contributory local factors. In accordance with this view, clinical investigations demonstrate that the antrum of Highmore holds an important position in the etiology of hay-fever and in some of the commoner forms of catarrhal and nervous disturbances of the nose. A series of a large number of cases coming under this class of affections, I

have studied during the past number of years with considerable interest. The results of my observations have been of such a character as to corroborate the theory.

To discuss empyema of the antrum in this connection is not the province of this report. In the first place, the design is to deal with non-suppurative processes. While of the pus-producing conditions of the antrum much has been said in recent years and medical literature abounds in valuable contributions on the subject. Reference will be made to suppurative maxillary sinusitis only for the purpose of illustrating pathologic and symptomatic relationships.

It is my belief that the nervous catarrhal manifestations of the nose, in their relation to the presence of a catarrhal maxillary sinusitis, are of sufficient importance to merit attention and suggest further investigation. Is it not logical to associate disturbances of the maxillary sinus with the various forms of nervous coryza, when it is remembered that an intimate anatomic and physiologic relationship exists between this cavity and the nasal passages in their nerve supply and mucous membrane connections?

In order to show the ground for believing that the antrum forms an etiologic factor in the causation of hay-fever, I will quote in this connection the history of my first case treated in a manner herein described:

F. N. D., aged 43 years, lawyer, suffered from hay-fever for a period of twenty-five years. Nine months before his first attack, he sustained an injury to the nose from a blow received on the right side over the region of the maxillary sinus. This injury was followed by stenosis of the left nostril, which at the time of my examination twelve years ago, when he first consulted me on account of hay-fever, was found to be obstructed by the presence of a deviation and exostosis of the septum. The patient contended that the hay-fever resulted from the traumatism, as prior to the infliction of the injury he experienced none of the symptoms peculiar to the disease. This occurred early in the spring, and the attack of hay-fever came on in the autumn of the same year.

At the time of our first interview (12 years ago) his suffering was intense and the symptoms of the disease were pronounced, chief among which was an orbital cellulitis of a serious character. With a view to lessen the severity of the symptoms, I suggested a correction of the septal deformity as soon as the urgent symptoms had sufficiently subsided to warrant operative interference.

Removal of the exostosis and correction of the deviation, improved his respiration and general condition; but he still continued to suffer thereafter more or less serevely from the disease during subsequent years. It was a common occurence for him to be incapacitated for work from six to eight weeks out of the year, and he was left at the end of each season in a state of general physical debility. In the latter part of December, 1903, he began to complain of facial neuralgia and severe pain over the right superior maxillary bone. He consulted me in reference to this trouble, and I found that the antral cavity was very much disturbed, as was evidenced by the presence of offensive discharges in the right nostril. The antrum was cleansed by irrigation through the opening communicating with the nose. A warm normal salt solution was used. The contents of the sinus as they appeared in the drainage basin, were found to be most offensive, the discharge consisting of inspissated muco-purulent material which had undergone fermentation and decomposition. A number of similar irrigations at different intervals succeeded in affording complete relief, both as to maxillary pain or tenderness and the condition of the sinus itself.

Of considerable interest was the fact that the opening of the antrum in this case was extremely abnormal, as it was possible to introduce a large-sized Eustachian catheter.

This unusual opening, without doubt, by virtue of its admitting readily into the sinus particles of extraneous irritating or infective substances, contributed in no small degree, toward creating the disturbance of which he was suffering at the time.

Having known the man so well as a hay-fever victim in previous years, it occurred to me that, perhaps, the diseased antrum and its abnormal opening, formed the basic lesion of his autumnal catarrh. The suspicion that this might be so, ended in a mutual understanding that when the next hay-fever season arrived, I should treat the antrum for experimental purposes. About the middle of August (1904) he was seized with the usual symptoms of the disease, but he did not visit me until two weeks later, when he recalled our mutual arrangement. During these two weeks he had suffered about the same as on former occasions, and on the day of his visit the symptoms were well marked. I washed out the antrum with a very warm solution of boric acid, the return fluid from the cavity containing quantities of retained and decomposed secretions; the syringing of the sinus was continued at each sitting until the

solution appeared clear in the catch basin. These irrigations were followed by injections of a warm 5 per cent solution of mentholated oil into the antrum.

The next day the symptoms had materially abated and I again treated him as on the day previous. He did not return until two weeks later, when he presented himself more for the purpose of making a report than to be treated. He said after the second treatment all the symptoms, both local and general, disappeared. In 1905, he gave the method a thorough trial. During the season, beginning August 15th, he was treated on five different occasions every other day and thereafter once a week, which treatment had the desired effect, and his suffering as compared with other years was minimized to such a degree as to render him absolutely comfortable. As the mentholated oil was rather pungent and painful, it was abandoned, and in lieu thereof *thymol iodide* was substituted and used in the form of an insufflation in the maxillary sinus it was cleansed with the boric acid solution, thereby rendering the treatment more tolerable and efficacious.

The past year (1906), for experimental purposes, the patient permitted me to apply the treatment occasionally, with a view to seeing whether or not the hay-fever season could be passed without the appearance of any of the symptoms. Once or twice a month during the year this was done, and it is interesting to note that last season (1906) the patient remained unaffected with but one exception, namely: one day he was making researches among old, stored-away, dusty legal books and pamphlets, when the exposure to the dust brought on an array of nasal symptoms simulating those of hay-fever, whereupon the patient immediately came to my office for relief and the antral cavity was subjected to the same procedure as before, with the gratifying result of at once eradicating the trouble. Further experimentations will be required to solve the problem of immunity as not only regards this case, but also others in which similar results were obtained. Just now it is too early to consider this question.

It will be noted, however, that I have spoken of the right antrum only; the left side having received no attention.

The patient's suspicion that the trauma twenty-five years ago brought on the hay-fever may have some foundation: the injury of the nose and maxillary bone evidently had the effect of impairing the integrity of the sinus and of the ostium as well, and the history

of the case and the results of treatment in a way strongly confirm this view.

The patient speaks of the treatment as follows:

"The result of these treatments has been surprising and most gratifying. I have passed through the hay-fever season without any annoyance whatever. I have lost not a moment's sleep. I have not been incapacitated for business in any way. I have attended the theater, taken long drives in the country, and long walks in the woods and fields, things not to be thought of in seasons past. In fact, I have escaped all annoyance and distress incident to hay-fever, and feel that hereafter I shall be at once relieved and need no longer dread the hay-fever season."

We have presented in this instance an example furnishing facts as related to hay-fever, that are worthy of recognition and serious reflection with regard to the etiology of the disease. The history of the case and the results of treatment almost conclusively demonstrate that the antrum was primarily at fault and formed, as it were, the basic lesion.

THE OSTIUM MAXILLARE.

The *pivotal point* of the antral theory of hay-fever is the ostium maxillare. The anatomic condition and location of the orifice of the antrum, determine the etiologic and pathologic features of the part played by the maxillary sinus in the production of hay-fever phenomena.

Generally speaking, the antrum of Highmore is practically a sterile cavity, particularly, so long as it remains uninvaded by active pathogenic germs, but when irritation or infection does obtain, it will be seen by investigation, that a supplementary or a malformed opening of the antrum exists, and fundamentally contributes to the sources of irritation or infection of the sinus.

Bacteriologists may say the antrum is not sterile, for the reason that various bacteria are always present in the nasal passages with which the sinus communicates. In the main this is true, but are they not inactive germs? When it is taken into account that the ostium maxillare in a strictly normal sense is of definite construction, and by virtue of its location has but one function, namely, that of supplying air to the antrum and guarding its integrity against the entrance of infective or irritating agents, it is altogether natural to consider it sterile, in spite of the presence of non-active bacteria.

As soon, however, as the ostium deviates in size and course from what nature designed it to be, then the antral cavity may at any time become subject to the action of morbific influences. Reasoning from analogy it can be argued, that, if the middle ear or cavum tympanum is practically sterile in the absence of germ activity, despite its communication through the Eustachian tube with the naso-pharynx, the maxillary sinus for like reason is entitled to the same consideration. Therefore, it would be seen that the ostium maxillare bears the same relation to the maxillary sinus as does the Eustachian tube to the cavum tympanum, and consequently to maintain normal integrity of the respective cavities, no alterations of the tubes or ducts should be present.

The normal opening of the antrum is intricately located with reference to anatomic landmarks, which are not constant and often make exploratory procedures difficult or impossible.

Situated in the middle meatus underneath the middle turbinated body and at the posterior end of the hiatus semilunaris, between the uncinate process and the bulla ethmoidalis, the opening of the antrum is more or less concealed, We can readily understand how undue development of the bulla and of the uncinate process may contract the semi-lunar hiatus and increase the amount of concealment, thus rendering the ostium absolutely inaccessible.

The normal ostium is about 2 mm. by 3 mm. and leads to the maxillary sinus in a direction downward, sharply forward and outward. It would seem that the obscure location of the ostium and its tortuous direction, so to speak, indicate nature's design with regard to the function of the opening. In my opinion it has but one function and that is to supply the antrum, a pneumatic cavity, with air. In a perfectly normal state it is very doubtful whether it was ever intended to serve the purpose of a drainage canal, its very location favors this opinion when viewed from a physiologic standpoint. The opening exists at the extreme upper part of the naso-antral wall in the region of the middle meatus of the nose where the inspired current of air reaches the channel and antral orifice in full volume, and thereby ventilates the antrum. This physiologic principle furnishes abundant reason for the location of the ostium, and it is only on abnormal or pathologic grounds that the orifice can be safely regarded as a drainage canal. The question is sometimes asked: How is it possible to wash out or irrigate the antrum through the normal opening? It is not possible to do so.

It is only practicable when the opening is abnormally large or when there exists a supplementary communication between the nose and the antrum. When the opening of the antrum deviates in its normal dimensions (2 mm. by 3 mm.) and departs from its natural course, it no longer sustains its primary relation, but becomes an avenue of communication, fraught with morbid influences productive of antral disturbances.

In certain instances, on dissection, the opening has been found to measure in its antero-posterior diameter as much as 18 mm. (Turner). Where so great a change, or even less, in the diameter of the ostium takes place, it may be concluded that the course of the duct is also changed and proceeds more directly toward the antrum than otherwise. This variation undoubtedly is due to anatomical defects or in cases of sclerosis of the nose to atrophic changes of the tissues.

There frequently exists a supplementary or accessory opening of the antrum and according to Zuckerkandl it may be present in 10 per cent of cases: Logan Turner found it to be present four times out of nine dissections, while Douglas says it occurs in 50 per cent of cases, and clinical observations confirm the statement of the last named authority. These statisticians are important and shed considerable light on the antral theory of hay-fever and catarrhal maxillary sinusitis.

The location of accessory ostia is usually above the inferior turbinated body at the junction of the middle with the posterior third of the bone on a line without and below the posterior end of the semilunar canal. They occur in the membranous portion of the naso-antral wall and range in size from the head of a pin to 8 mm., and sometimes more than one exists on the same side; but whether these accessory openings are formed during the developmental period of the antrum or whether they are mainly due to disease, or trauma, is not fully understood.

Taking into consideration their varying dimensions and the frequency with which they occur in a given series of cases, these supplementary openings of the antrum form a conspicuous part played by the maxillary sinus in the production of the disease under consideration. Further than this, they occupy a favorable position in the middle meatus for exploratory and therapeutic purposes. In the presence of these anatomic naso-antral irregularities, so far as they pertain to the orifice of the antrum, it is not difficult to comprehend

the *modus operandi* of the various extraneous exciting causes peculiar to the induction of hay-fever, and for the same reason it is not hard to understand how a catarrhal maxillary sinusitis produces hypersecretion of the cavity and in turn provokes nasal irritation.

A comparative study shows that as to size the antrum varies in different individuals. It is lined by mucous membrane consisting of ciliated epithelium and a rich distribution of muciparous glands, and it is in part, at least, analogous to that of the nasal fossae.

NERVOUS CONNECTIONS.

The antral mucosa receives numerous sensitive nervous filaments given off by the superior maxillary division of the trigeminus, and it is also abundantly supplied by secretory nervous fibers.

The anterior dental, the posterior dental and the superior nasal from Meckel's ganglion, form the nervous supply of the antrum (Gray). Sympathetic fibers come from the Vidian, composed of the great petrosal and carotid. The carotid forms a connection between the carotid plexus of the sympathetic and Meckel's ganglion. Nervous filaments are given off from the lower border of the posterior dental which form a minute plexus on the outer wall of the superior maxillary bone immediately above the alveoli. From this plexus, filaments are distributed to pulps of molar and second bicuspid teeth, *the lining membrane of the antrum* and a corresponding portion of the gums. The anterior dental is given off just before its exit from the infraorbital foramen; it enters a special canal in the anterior wall of the antrum and anastomoses with the posterior dental and superior nasal branches. From this nerve some filaments are distributed to the incisor, canine and first bicuspid; others are lost upon the lining membrane covering the fore part of the inferior meatus. In this situation it forms a connection with a nasal branch from Meckel's ganglion called the ganglion of Bochdalek.

From Meckel's ganglion are branches (descending or palatine) supplying the lining membrane of the nose. It has three sets of fibres; motor, sensory and sympathetic. Internal branches are distributed to the septum, and outer wall of the nasal fossa; one branch (posterior) is distributed to the outer wall of the antrum and forms a communication with the anterior dental in the ganglion of Bochdalek.

From this we see the antrum to be abundantly supplied with nerves. The posterior dental, anterior dental and the branches

from Meckel's ganglion give motor, sensory and sympathetic not less to the antrum than to the meati of the nose. Irritation of these fibres in the antrum is not only capable of producing the sensory and sympathetic disturbances seen in hay-fever, but neglect to relieve the antrum from sources of irritation and disease, explains the discouraging failure to cure the condition by operative and other therapy upon the nasal cavities. Moreover, the continued irritation of these sensory and sympathetic fibres is capable of establishing in those of unstable nervous organization that distressing chain of neurasthenic symptoms which are the secondary and coincident conditions in most cases, for under emotional and morbific influences a reciprocal relationship between the sympathetic and central nervous system exists.

In hay-fever the sensitive nerve endings, found present in the antral and rhinal cavities, are kept in a state of excitation as a result of local irritation.

PATHOLOGIC CONNECTIONS.

The maxillary sinus, apart from its orifice, is a closed cavity where warmth and moisture have ample chance to engender fermentation and generate bacterial products. Its mucous membrane is continuous with that of the nasal passages through the ostium maxillare. This arrangement, under abnormal anatomic conditions, with regard to the naso-antral orifice, forms the pathologic connections between the antrum and the nose, and shows how readily an inflammation of the antrum can reflect itself to the nose by extension or by irritation consequent to its discharging contents. In hay-fever, the secretions arising from inflammation of the antrum, are the source of the vaso-motor nasal disturbances and the associated reflex symptoms, which are relatively augmented or diminished in proportion to the quantity and irritative properties of those discharges.

The query arises, how does the antrum get rid of its fluid contents? In the first place, gravity performs an important part in the function. Various positions or movements of the head favor the exit of the secretions. In the second place, it is demonstrable that the act of respiration or of blowing the nose, drains the cavity by a process of suction. The peculiarly favorable position of the accessory or malformed ostium, as it is related to the middle meatus and the middle and upper turbinated bodies, make drainage possible in the manner described.

I have frequently observed that after washing out the sinus for therapeutic purposes, the fluid was thoroughly evacuated by the time the patient had finished blowing the nose, as was illustrated by the use of the air bag and insufflation of medicinal powders.

With regard to the so-called extraneous exciting causes of hay-fever, such as pollen from weeds, grasses and flowers, odoriferous emanations, dust, certain drugs, etc., etc., how are they related to the antral theory of the disease? Is it not possible and is it not a fact, that these external excitants, after reaching the nasal passages, enter the antral cavity and through their action induce hyperemia and catarrhal inflammation productive of hypersecretion and, in case of chronic pre-existing antral disease, intensify the activity of the lesion?

I am of the opinion that when the communication between the nose and antrum is absolutely anatomically normal as to size and situation, disturbances of the character mentioned are not likely to take place in the absence of alveolar or dental irritation, but in instances where the ostium is abnormally large, or where there is an accessory opening, an avenue is created for the favorable entrance into the cavity of extraneous irritative or infective substances by air in motion or the act of respiration. Thus we can readily see how these foreign substances can enter and lodge in the antrum, induce irritation and infection, and give rise to inflammation which usually is secretory in character.

Fundamentally speaking, the whole theory rests on anatomic abnormalities.

In 1904, Emanuel Fink, Hamburg, Germany, in a contribution, drew attention to the antrum of Highmore as furnishing the probable source of the disease, and successfully treated it in accordance with these views. In support of the antral theory Fink quotes Helmholtz, who himself was a sufferer from the disorder, and ascribed the affection to the influences of certain vibrios contained in the secretion of the nose thrown off by violent attacks of sneezing, while on the other hand Helmholtz contended that a secretion which passed from the nasal passages, drop by drop, did not present the vibrios; they were lodged in the variously hidden recesses of the sinuses of the nose. It is maintained that even if the vibrios could not be regarded as a specific form of disease, Helmholtz's observations would prove that the secretion must come from the sinuses, especially from the antrum of Highmore. Sneezing, a

constant accompaniment of hay-fever, is, according to Sandmann, the result of irritation of the ethmoidalis nerve. This irritation may be caused either directly through the agents contained in the inspired air, or indirectly through the secretion flowing from the antrum.

Concerning asthma, it has been shown by reliable authority (Francois, France and Lazarus), that through excitement of the nasal mucous membrane, spasm of the bronchial muscles can be produced reflexly. Recent physiologic researches show that the mucous membrane of the antrum of Highmore when disturbed stands in the same relation to bronchial spasm as does that of the nasal passages.

It is no longer questioned that a number of respiratory, as well as associated diseases, are both directly and indirectly due to sinus affections; for instance the source of nasal polypi, headache, ophthalmic disorders, chronic rhinitis (atrophic in particular), mental derangements, trifacial neuralgia, odontalgia, bronchial asthma, etc., etc., is often capable of such demonstration by operative and other measures.

NEURASTHENIC ASPECTS.

Earlier as well as more modern writers maintain that hay-fever is a neurosis and in display of symptoms perhaps it may be true, as the nervous characteristics of the disease largely forms the symptomatology and in fact, it is this feature of the affection that gives rise to the troublesome complications with which the physician is called upon to contend. Do the nervous manifestations constitute the disease, per se, without the existence of a primary lesion? It is hardly logical to assume such a position, when it is known that pathologic states involving other organs of the body are often responsible for the presence of neurasthenic symptoms.

When the late Dr. Geo. M. Beard introduced into our medical nomenclature the term neurasthenia, he did not intend that the use of the name should be applied to diseased processes, inducing a group of peculiar nervous symptoms hitherto misunderstood, for the purpose of disguising erroneous diagnoses or concealing sins of omission and commission. He maintained that these neurotic symptoms sprang from definite, tangible causes and that by grouping them together under one general designation he thereby would give them a special significance; for instance, when they accompanied a gastric disease, it was termed "gastric neurasthenia," and

when associated with a disturbance or abuse of the sexual organs, "sexual neurasthenia," and so on. Therefore, with the same propriety, the neurotic phenomona attending nasal nervous catarrh, due to sinus origin, may be called *sinus* neurasthenia. There has always been a tendency to emphasize the importance of the nervous symptoms of hay-fever; this is exemplified by the many different forms of treatments adopted with a view to meet symptom emergencies only, while its organic phase is lost sight of or not inquired into.

There are those also who contend that the nervous features are partly imaginary. Few ailments of the body can justly be ascribed to the power of the imagination alone. Under extraordinary circumstances, perhaps, such a belief is sometimes tenable, especially when hysteria is present, but it is not infrequent, however, that on closer scrutiny of existing conditions as to origin, a hidden morbid state, regardless of its location, is often found in given cases to be at the bottom of the hysteria.

In hay-fever subjects, where a nervous state of the individual predominates, causal factors ought always to be considered, because if the neurasthenia is a secondary factor, and simply constitutes a reflection of disturbed nervous harmony or equilibrium, induced by distinct lesions acting directly or indirectly on an unstable central nervous system, the solution of the problem becomes easier. It is also true that certain basic conditions of disease are capable of creating in patients of nervous instability, a special susceptibility to exciting causes. These persons are those of idiosyncratic temperament, so-called. I believe this to be the case in hay-fever, whose phenomena are ascribed by some authors to a special idiosyncracy. In other words, the idiosyncracy is the result of morbid central evolutionary processes, in which the sympathetic and vaso-motor nervous system play a conspicuous part in establishing the hay-fever predisposition. External excitants are only contributory factors and hold a subordinate position in etiology, and excite into action a lesion over which they probably exercise an influence. Internal disease must be present in order to enable impressions to exert their effects on the central nervous system.

Hay-fever belongs to this category of neuroses. It is a general observation that diseases, hampered by obscurity respecting their fundamental sources, always make rational therapeutics difficult; for this reason the physician is not infrequently called upon to make

an uncertain diagnosis and employ a symptomatic therapy in order to satisfy the patient, and in so doing he himself unfortunately becomes satisfied and makes no further research. From the patient's point of view and demands, not a few symptoms of the disease require special attention, thus increasing the number of remedies employed for purposes of alleviaton. A multiplicity of remedies will, however, become unnecessary, when we once fully understand the source of the disease and adapt our remedial measures accordingly and pay less attention to individual symptoms.

With reference to the so-called sensitive areas of the nose acting as etiologic factors of hay-fever, anatomically considered, these areas consist of a localized distribution of sensitive nervous filaments in certain parts of the nasal mucous membrane. Under local stimulation or irritation they are readily excited; they serve both physiologic and pathologic purposes and their degree of sensitiveness normally varies in different individuals. Physiologically they serve as safeguards to the upper air tract, but on the other hand, by oft-repeated and prolonged local irritation, the sensitiveness increases with a resulting hyperesthesia, which in turn becomes a pathologic condition. Hyperesthesia of the nasal passages thus appearing is prone to lead to functional derangement of the sympathetic nervous system, and induce reflex symptoms and vaso-motor disorders, such as coughing, sneezing, asthma, nasal fullness and discharges, tinnitus aurium, etc., etc. Though characterized by a neurotic physiognomy, it is neverthless contended that the disease may be primarily organic, and that a pathologic state of the antrum of Highmore, so far as my present observations and experiments extend, is the seat of the chief etiological lesion. Personal experiences demonstrate the theory in a practical way and set aside the illogical conclusion, that the neurasthenia constiutes the malady. Errors are sometimes committed by hastily formed conclusions with regard to the immediate cause of a sudden onset of the disease, as is instanced by the celebrated case of "rose cold," or hyperaesthetic rhinitis, which was presumably produced by the presence of an artificial rose, reported some years since, and which account is still fresh in the minds of many rhinologists. This singular circumstance will continue to be referred to whenever discussions arise with reference to the causation of hay-fever. Is it not possible that the artificial rose served only a coincident circumstance peculiar to the situation during the patient's visit? The history of the case is not sufficient

to strengthen the argument in favor of "suggestion" as related to hay-fever, because a series of cases similarly affected would be required to give scientific value to the observation.

The will has a definite power over the operations of the body both in health and disease, but psychic influence, however, must not be over-estimated in its control over the physical organization. It no doubt serves a valuable purpose under extreme circumstances, as when faith in what is being done for the relief of suffering is exercised, because then an element of expectancy is introduced into the treatment, and the mind is thereby put at rest, and nutrition of the nervous system is re-established and physical forces re-assert themselves. This we see illustrated sometimes in physical ailments as well as in mental conditions of grief, fear or undue anxiety. As applied to the induction of organic disease, psychic doctrines should not be too enthusiastically considered in these days of scientific medicine, as their supposed miraculous power in great measure is a relic of past ages. In the days when medical empiricism prevailed and the physician had no pathology and only a meager etiology to govern him in the selection of his remedies, there was some color of reason for such doctrines.

In the consideration of the alleged mental aspect of the disease the foregoing statements have been made, because hay-fever is not a disease of the mind; it is a disease under the domain of cause and effect, and in all probability, due to an organic lesion in which the antrum of Highmore is an important contributory factor.

DESCRIPTION OF CASES.

As the limits of this paper will not permit a detailed account of each and every case under observation and treatment during the past three years, I will confine myself to description of salient points developed in the experience in order to illustrate as specifically as possible the intimate connection between the antrum and hay-fever.

In the period mentioned there were under my observation ninety-one cases of hay-fever and rose-cold in varying degrees; two were treated in 1904, fifteen in 1905, and seventy-four in 1906, with uniformly good results.

To justify myself against probable criticism, I said to each new patient, that while I firmly believed the treatment logical, yet the method was an experiment, and, apart from safety, no promises would be made as to outcome.

The results realized during the first two years were sufficiently satisfactory to encourage further investigations in a more extended manner, wherefore in the past year an especial effort on the part of some of my professional friends and myself, was made to bring under observation as many instances of the disease as was expedient, whereby the gist of the theory could be verified.

Case: Accessory Ostia and Typical Symptoms. Mrs. B., aged 33 years, wife of a physician and mother of eight children, consulted me about eighteen months ago, with reference to hay-fever. She was of a lithemic diathesis and suffered at times from inter-costal and facial neuralgia. She contracted the disease in August, 1895, and has been a yearly sufferer since then with progressively greater severity; a dry, dusty day, a shopping tour or smoke, sufficing to bring on an attack; the most troublesome symptoms being sneezing, rhinorrhoea, pain in the nose, and asthma. At the height of the attack there were usually present excessive lachrimation, burning and itching sensations of the eyes.

Prior to her consultation with me, she had tried various remedies, such as Dunbar serum, adrenalin, cocain, antilithemics and many others; and besides these, intranasal surgery was employed for the reduction of hypertrophic and other irregularities of the nose, with the hope of overcoming the affection; all, however, without obtaining relief.

Examination of the nose revealed an intensely red, swollen mucous membrane of both sides, which was very sensitive, as evidenced by violent reflex disturbances when the sound was used.

The middle meatus on the left side around the orifice of the antrum was engorged and hypersensitive, whereas the right side in this respect was not involved to the same degree.

After reducing the sensitive and swollen membrane by applications of cocain and adrenalin, thereby improving the local field of vision, I found by sounding that the opening of the antrum was supplementary and very large, and therefore concluded that this case came within the purview of the antral theory, and about the latter part of that season treated her accordingly.

After the first treatment, the eye, throat, chest and severity of the nasal symptoms practically disappeared within twenty-four hours, and it is interesting to note that the patient's first night was passed comfortably, and upon a second application, within a very short time, all the signs of the disease were dissipated.

Prior to the treatments she was unable to attend theaters, go shopping, or come in contact with hot winds and dust, or drive where exposed to weeds, without suffering discomfort or being compelled to remain in her room with closed doors and windows, and in order to test the theory, the patient attended the theaters, went shopping and withstood the action of the elements and contamination of weeds, with the result, that she slept well, felt no discomfort therefrom and her asthma entirely left her.

The malformed ostia favored instrumentation as it was quite possible to introduce the canula of the syringe or powder blower into the antrum. Applications of a warm solution of boric acid in the form of injections, removed from both sinuses an unusual amount of retained secretions, suggesting inflammation and hypersecretion of the antral mucous membrane. Mrs. B. continued well until house-cleaning time arrived, when she was exposed to dust and odors arising from new grass-matting on the floors—an environment which in the past was always accompanied by an array of hay-fever symptoms, regardless of the time of year, thus showing that some certain organ associated with the nasal air-tract was implicated in the morbid process and formed the basis of disturbance on these occasions, owing to irritants acting from without.

Between the season of 1905 and 1906 at the suggestion of her husband, a physician, Mrs. B. visited me at stated intervals for purposes of treatment to see whether the opinion that the maxillary sinuses were organically involved in the production of these symptoms could be verified. The antra were regularly cleansed at intervals and afterwards medicated with an application of thymol iodide or a 25 per cent solution of argyrol.

Thereafter she frequently tested herself and found that she could expose herself to irritants, dust and odors, with impunity.

The past hay-fever season went by without much trouble, only one attack lasting about four days manifested itself, further treatment at this time being impossible owing to the delicate condition of the patient. She has since declared herself well and is perfectly able to go about and attend to her usual duties.

CASE: ASTHMA.

September, 1905, Mr. W., aged 40 years, a furrier, constantly exposed to dust and odor peculiar to his occupation, was referred to me on account of asthma contracted two and one-half years previously and which from its inception has increased in intensity day

and night. Upon careful examination, beyond a periodic discharge from the nose, I was unable to find the presence of the usual lesions of the nose which would account for the *paroxysms*. There were no intranasal deformities or pressure contact and the only suggestion that the nose might have been responsible for the attacks, was an intumescent thickening of the mucous membrance of the septum directly opposite the middle turbinate bodies, which feature was taken into consideration as a causative factor.

I was of the opinion that the case was one of bronchial asthma, produced by the inhalation of fur dust, and with this view he was advised to keep away from his occupation, be exposed to the open air as much as possible, exercise judiciously and take the medicine prescribed. Iodide of potassium alone, as well as in mixtures containing other anti-asthmatic agents, was faithfully tried without avail, and in fact almost the whole category of remedies recommended for the cure of asthma, was resorted to in the course of time, with negative results. Concluding that constitutional treatment was a failure, the patient was given local treatment, accompanied by intra-bronchial injections of warm mentholated oil, and while this procedure yielded partial relief it did not cure him. The hypertrophied tissue on the septum opposite the middle turbinates was then cauterized; but without results, and, as I was at this time directing my attention to the maxillary antra of patients afflicted with hay-fever, it occurred to me, that perhaps his sinuses were affected by the entrance of dust or other infection arising from the character of his business and that similar treatment might dissipate the asthma. I treated him accordingly and it was highly gratifying. I found that the same substance came from the cavities as did from hay-fever subjects, that is, a flaky-white or milk-curdle deposit.

Upon receiving a visit from the man the following day he greeted me with, "You struck it this time. I had no asthma last night." At stated intervals, he was treated six or eight times.

At present writing the patient is free from asthma and, after obtaining relief, resumed his business and has been thus engaged ever since. I feel confident he will remain permanently free until such time, at least, as the conditions of his environment may again disturb the sensitive nerve supply and mucous lining of the maxillary cavities.

NEURASTHENIA AND NERVOUS NASAL CATARRH.

On June 18th, 1906, Mrs. A., aged 40 years, the mother of four children, for a number of years was afflicted with the hysterical type of neurasthenia manifesting itself in various ways. After a long continued course of treatment by a gynecologist who entertained the opinion that the nervous symptoms were due to a diseased state of the pelvic organs, a nervous disturbance of the nose developed. Later on hyperaesthesia of the skin of the right side of the face, extending down and over the shoulder and arm of the same side, appeared. The throat and tongue became involved, and she at first imagined that an apple seed had buried itself in the tissues of the right side of the throat and formed a focus of disturbance. These symptoms were accompanied by burning and itching of the mucous membrane of the eyes, mouth, tongue and throat, which still existed when she consulted me. There were present frontal and occipital headache, frequent paroxysms of sneezing and excessive discharges of the nose, restless nights and loss of appetite. Evidences of impaired nutrition and a feeling of melancholia, which so often accompanies these conditions, existed in a marked degree. Coupling the signs of nervous nasal catarrh with these general nervous manifestations, it occurred to me that the chief causal factor of the complaint was of sinus origin.

On examination of the nose I found turbinal hypertrophy, hyperaesthesia and soggy appearance of the mucous membrane. The report of this case is not presented as one of genuine hay-fever. It is offered as an admirable illustration of a type of neurotic affections of the nose known as nervous coryza, characterized by a significant display of neurasthenic phenomena strongly resembling the symptoms of hay-fever and coming from the same organic lesion located in the antrum of Highmore. Sounding the maxillary sinuses with a probe developed painful sensations in the middle meati. In fact, the whole interior of the passages of the nose was hyperaesthetic and made the use of the instrument almost unbearable. In order to reduce the swollen mucous membrane and render instrumentation of the sinuses possible, adrenalin and cocain were applied.

The cavities were cleaned with a syringe and warm boric acid lotion. From the left cavity came a fluid (four drachms in quantity) resembling butter-milk, while the right discharged a sero-mucus secretion. An interesting circumstance in connection with

the case is that, about fifteen years ago, Mrs. A. was struck on the right side of the nose and in consequence suffered total loss of the sense of smell as well as periodic attacks of intense facial neuralgia and pain in her upper teeth. Percussion over the cheek in the region of the antra elicited pain in the deeper structures. The patient was regularly treated for about two weeks and it is interesting to note that the sense of smell has returned in an exaggerated form; the nasal, head and throat symptoms, as well as the hyperaesthesia of the skin, are dissipated and the improvement in every way is marked. She is now able to sleep all night; her appetite has returned; her mentality is changed, and the neurasthenic phase of the case have disappeared. No medicines except those used in the local treatment were prescribed.

TEST CASE.

One man in particular, Mr. D., capitalist, consulted me seven days before the date of his periodical attacks of hay-fever, which always had been on the 7th of August, to find out whether or not something could be done to prevent his having the disease. He informed me that he suffered severely for many years and that it rendered him perfectly miserable and melancholy for at least two months during each season. In fact, his mental condition was of such a character as to cause him to stay in the attic of his residence day and night in order to get away from, fancied, atmospheric influences of hay-fever. As he was urgently advised to see me by Mr. D., whose case is quoted at the beginning of this article, he came not wholly without confidence. Examination showed large accessory ostia on both sides and the maxillary sinuses catarrhally affected.

The malplaced ostium and the readiness with which it could be entered, made me feel that the case was highly amenable to treatment, and I so expressed myself to him. Treatment was begun on the same day of the examination and continued daily for some time thereafter for the reason that he looked upon it as a luxury.

Results: The 7th day of August arrived, and no sign of hay-fever; the 8th, the 9th, and the 10th, were likewise negative. About the 11th, out of curiosity, he tested himself by making a tour of one hundred miles with an automobile through the country where all the elements peculiar to the exciting causes of the disease existed. The next day he reported his experience and, aside from itching at the end of the nose, he made no complaint. Upon wash-

ing out the antra, however, I was amazed at the quantity of milk-curdle secretions that appeared in the catch basin. This evidenced valuable information with regard to the fact that the antra were undergoing an inflammatory disturbance and in a brief length of time would have ushered in an array of hay-fever symptoms through nerve irritation; insufflation of thymol iodide however arrested the process and no trouble came from the exposure.

For a further test this same experiment was resorted to a number of times during the season but nothing came from it to interrupt the comfort of the patient, except an occasional itching of the end of the nose.

ANTRA VS. DUNBAR TREATMENT.

Mr. S., aged 45 years, teacher, a victim of hay-fever for seven years; symptoms, sneezing, nasal discharges, coughing and severe paroxysms of asthma. He first visited me August 10th, 1905, at which time the Dunbar serum was prescribed in powder form and was continued until September 6th, without effecting any benefit, and during this visit the antral method was recommended and applied with the result that the asthma disappeared within the first twenty-four hours, and, after several subsequent treatments, and within a week, he was able to follow his profession without any inconvenience and considered himself practically cured.

At the beginning of the hay-fever season and about the first of August, 1906, while upon a business trip through Ohio, he was taken seriously ill with the disease and upon his arrival at his destination was again given the Dunbar serum treatment, which was continued for about ten days until his arrival in St. Paul, when he presented himself at my office. I found him suffering from his former usual symptoms and asthma in particular. Upon request of the patient I again applied the antral treatment, with the result that within one week he enjoyed perfect relief.

LARGE SUPPLEMENTARY OSTIA, DEEP ANTRAL CAVITIES AND DECIDED NEURASTHENIA.

Dr. W., aged 32 years, neurotic temperament, good family history, came to me September last, complaining of neurasthenia, restless nights, stenosis of the nose, paroxysms of sneezing, pain and congestion of the eyes, asthma and profuse discharges from the anterior and poterior nares; these symptoms, without doubt, denoted an attack of hay-fever.

Ten years prior, he experienced his first attack and suffered thereafter more or less severely. During this time surgical procedures brought about beneficial results, but as time elapsed their good effects gradually disappeared, as dampness, drafts, dust, over-heated rooms, exposure of extremities while asleep and decided weather changes always induced recurrence of the symptoms, regardless of the time of year, the symptoms intensifying themselves only during the hay-fever period, whereby the suffering was augmented.

Four years ago the patient came to me afflicted with one of these violent seizures and I prescribed the usual palliative remedies then in vogue, but accomplished nothing beyond temporary relief. On seeing him this past season he was suffering unbearable tortures and the status of the case at this time was as outlined above.

He went East for the purpose of consulting with medical authorities regarding his case and after being informed that his symptoms were largely imaginary, he returned to the West dissatisfied and discouraged with his Eastern experiences, and visited my office ostensibly for antral investigation and treatment.

On examination I found the nasal passages unusually irritable and engorged, the mucous membrane being soggy and edematous. After intranasal sensitiveness and engorgement had been reduced by applications of cocain and adrenalin, it became possible to explore the location and character of the maxillary openings. They were found to be supplementary, very large and readily sounded, and occupied a position in the membranous part of the naso-antral wall rather near the upper border of the inferior turbinated body. It was ascertained that both maxillary sinuses were of unusual size and had a capacity of six or eight drachms.

The antrum on both sides was first washed out with a normal salt-solution and as the retained secretions proceeding from the cavity, mixed with the injected solution, appeared in the catch basin, they presented a reddish, muddy and coffee grounds aspect. Because of the unusual size of the antra, a certain percentage of the injected fluid was retained, which made it necessary to use the syringe in order to withdraw the remaining contents which resembled what was caught in the basin. This irrigating process was continued until the sinuses were thoroughly cleansed. Injections of a 25 per cent solution of argyrol were then made and the patient was requested to lie down and keep quiet for an interval of an hour so

as to give the argyrol a chance to remain in contact with the antral mucous membrane in order to accomplish its ultimate purpose.

In this manner Dr. W. was treated for a period of a week at intervals of a day, each subsequent visitation evidencing improvement. Among the first symptoms to subside was the excessive nasopharyngeal discharge, which always gave much annoyance and interrupted the comfort of the patient at night when in the recumbent position. Next to disappear was the asthma, and at the end of the week the symptoms were entirely absent and the patient considered himself totally cured. In order to prove the permanency of the results he afterwards took frequent drives in the country and exposed himself to the exciting causes of the malady without recurrence of any of the symptoms.

Chiefly to be considered in this instance, in their bearing on the periodic nervous manifestations, are the various contributory external causes, the accessory openings and capaciousness of the maxillary sinuses, the high degree of antral inflammation and the character of the hyper-secretion of the cavities.

PRE-EXISTING CHRONIC CATARRHAL MAXILLARY SINUSITIS AND HYPERÆSTHETIC RHINITIS.

On October 29th, 1905, a man aged 46, contractor, consulted me relative to excessive catarrhal discharges from the nose, persistent coughing and retching spells, regularly present in the mornings and usually after breakfast, dyspeptic symptoms with loss of appetite, and consequent decrease of body weight. This state of physical impairment existed for a number of years without apparent cessation. Twelve years prior he was attacked by vertiginous seizures, the dizziness lasting for long periods at a time. The vertigo was thought by his physician to have been of stomachic origin.

On examination of the nose and postnasal cavities I found the former more or less occluded by turbinal hypertrophies and the latter occupied by disagreeable, tenacious secretions. A musty odor emanating from the air passages led me to believe that the so-called foul breath was due to the furred tongue and general gastric derangement.

With the idea in mind that perhaps the obstructed nose had a great deal to do with the symptom for which he chiefly sought relief, I reduced the intranasal hyperplasia which made nasal respiration comfortable. The postnasal space was treated by local applications of iodine and nitrate of silver pigments, used interchangeably.

This therapy in a manner afforded some benefit, but not enough to either satisfy myself or the patient. The morning retching continued and the offensive odor remained undiminished.

During August last, after a long interval between visits, he again saw me with the statement that he had a severe "cold in the head," as was manifested by nasal stenosis, sneezing attacks, watery discharges from the nose, itching and burning sensations of the eyes and throat, and on inspection of the nose and taking into account the symptoms presenting, I diagnosed it a clear case of hay-fever.

Inquiry developed that he had had similar attacks in previous years, but he supposed they were due to catching cold. At the time he came under my observation on this occasion, I was experimenting on the antrum of Highmore in other cases of hay-fever with regard to the relationship existing between the disease and the maxillary sinuses, ostensibly with the object in view to *prove* or *disprove* the correctness of the theory. The conclusion irresistibly impressed me that this case offered an excellent example for the purpose. Sounding the antral cavities demonstrated the practicability of entering them through the naso-antral opening with suitable instruments.

It was discovered by the method employed that an opening or communication of the antrum in both nasal chambers was present in the middle meatus at a point posterior and inferior to the meatal termination of the hiatus semilunaris; these openings proved to be accessory ostia.

This anatomic malformation, both as to position and size of the opening, a condition frequently present in hay-fever subjects, gave rise to the suspicion that the maxillary sinuses were diseased and formed the basic lesion of the patient's trouble. Both cavities were washed out with a normal salt solution, the irrigation being continued until the return fluid as it appeared in the catch vessel was entirely clear and free from sediment or retained secretions. The discharges thus forced from the antra were not exactly characteristic of what is usually noted in hay-fever conditions; it was offensive in odor and peculiarly muddy and granular in appearance, intermixed with mucoid material. A bacteriologic examination of a specimen revealed a green bacillus, *pyocyaneus*.

Six or seven consecutive treatments directed to both antra at intervals of twenty-four hours, had the desired effect in curing the patient of his hay-fever. He grew fond of the treatments because

of the good systemic effect produced and after the hay-fever had disappeared, he wanted the treatment often and regularly. It was not long until he remarked that the coughing, vomiting and retching had wholly disappeared as well as the postnasal discharges, his appetite and digestion at once improved, his weight increased, and the offensive odor was also brought under control and eradicated.

A point of particular interest, so far as subjective signs are concerned in antral disturbances of this sort, is that these patients usually know when a storm is brewing in the antral cavities, as is recognized by a disagreeable, burning sensation in the region of the middle meatus of the nose and in the neighborhood of the ostium.

This patient is always cognizant of what is going on at these periods and comes for a cleansing of the sinuses just as soon as the nasal sensations indicate the necessity, and one treatment usually suffices for a number of weeks. Catching cold or exposure to dust are the chief exciting causes in his case.

The deductions are—*first,* that the opinion is substantiated that one or more of the associated sinuses of the nose when in a diseased catarrhal state, is as a rule one of the chief causes of so-called post-nasal catarrh and its attending symptoms; *second,* that the history of the case furnishes a convincing argument in favor of the antral theory of hay-fever; and, *third,* that an abnormally large or an accessory maxillary ostium is a local anatomic predisposing factor in the etiology of catarrh and hypersecretion of the sinus.

GENERAL REMARKS AND DEDUCTIONS.

The observation is borne out by this experience that in those cases where unusually developed abnormal openings of the antra prevailed, the results of treatment were more prompt and satisfactory than in those where the abnormal dimensions of the communications were much less; the former favoring and facilitating instrumentation and the application of remedies, and the latter increasing the difficulties. The observance of this point offers a rational explanation on anatomic grounds for therapeutic failures.

Dissections have shown that the antrum of Highmore is a variable cavity as to its dimensions in different individuals, and also that an anomaly may sometimes exist, particularly as regards subdivision of the sinus into compartments by bony walls or partitions.

It is also observed that "rose-cold" or "hay-fever" of one or two season's existence in certain individuals, yielded, on an average, to

treatment more rapidly than it did in those who had had the disease for many seasons or where it extended over a long period of time. It may be concluded from this that as yet the antral mucous membrane had not undergone permanent inflammatory degeneration or changes in those patients who thus were readily cured. If this observation can be further verified in the future, it certainly adds a valuable factor to prognosis.

Of the whole number of cases heretofore referred to, one was a total failure. Though treatment directed to the antrum was faithfully followed for a reasonable period, yet no impression could be made and the symptoms refused to yield. The ostia were abnormal in size and no difficulty was encountered in the use of the probe or canula, but it seemed that whenever a fluid was injected or a powder blown into the antrum, it was found to rebound instantly into the nasal passage. Experimenting in different ways with a view of forming some explanation for the presence of an impediment, it was finally thought that the sinus was anomalous, there evidently being present a number of cavernous compartments. In given cases therefore of catarrhal maxillary sinusitis or hay-fever, in which local malformations obtain with regard to the location and size of the opening of the antrum, anomaly of the sinus must also be taken into account when dealing with a certain class where insurmountable barriers to treatment arise.

Also of this number three did not respond for lack of adequate treatment. Among the number there were twelve who were entirely relieved within a period of about two and one-half weeks, who had suffered in former years from six to eight weeks each season; the remainder were fully and completely relieved of the disease and its symptoms in a week or ten days; and, in exceptional instances, some in only four days; treatment of the last two classes having been administered daily.

My observation has been that the reflex symptoms first to disappear were, burning and other disturbances of the eyes, pruritus of the throat and ears, finally sneezing and discharges from the nose, and last *asthma* and cough.

That chronic catarrhal maxillary sinusitis is a morbid process productive of intranasal and postnasal catarrhal disturbances, is not to be questioned when it is understood that, by directing treatment to the antrum, many of these cases are relieved and get well. The

history of the last case quoted in this paper is an illustration and corroborates the opinion, and other instances coming under this same class can be referred to in detail as proving the fact.

For bacteriologic investigations specimens of retained secretions were taken *directly* from the maxillary sinuses of a certain type of cases and it was found that the green bacillus, *pyocyaneus*, was present in 65 per cent of the number. While this indicates nothing conclusive with regard to the role played by the "pyocyaneus" bacillus, yet it suggests further inquiry in this connection.

It may be questioned whether the antrum alone is engaged in the sinus process of the origin of hay-fever? May not the other associated sinuses of the nose be likewise implicated, an opinion entertained by Richards? Personal observations do not as yet prepare me to say that such is the case, however rational the opinion may seem. Principles of differentiations must be followed to establish the fact before conclusions can be arrived at with regard thereto.

Will antral therapy prove productive of immunity? In a certain class perhaps immunity can be attained, provided a systematic therapy be applied to the antrum during the intervals between seasons; especially so in cases where degeneration of the mucosa is not advanced or intractable and where anatomic deviations are pronounced and advantageous with reference to the opening of the antrum.

Then again, in instances where the antral cavity can not be reached via a naso-antral communication, one would be justified in making an artificial opening with trocar and canula into the sinus underneath the lower turbinate body for therapeutic purposes. Recently I adopted this method for the treatment of catarrhal maxillary sinusitis in three cases with excellent results following.

Considerable strength is added to the argument by results attained from treatment, that, in instances of asthma of antral origin, the irritation productive of the bronchial spasm primarily takes place in the antrum. As has already been referred to in the description of the nervous circle involved in the creation of central nervous instability, reflex and vasomotor disturbances of the upper air-tract, the mucous lining of the maxillary sinus is abundantly supplied by sensitive nerve endings and secretory nervous fibres to render it possible for such a phenomenon to occur and give rise to the asthmatic storm destined to follow.

It is not intended that the above statement should convey the idea that all cases of bronchial asthma organically arise from the antrum, but the opinion is clinically substantiated that not a few come from this source and that when all other means are exhausted as to cause and treatment, the antrum of Highmore should always be interrogated before one's efforts should cease in ascertaining the foundation of the disorder. Within the past month only, and while this contribution was in course of preparation, a man whom I had previously seen in attacks of hay-fever asthma, consulted me in reference to an obstinate and disabling attack of asthma from which he was then suffering. I found the naso-antral membranous wall of the left side of the nose badly disintegrated and for the greater part, absent, thus forming an opening of unusual size communicating with the sinus. No difficulty was experienced in inserting into the cavity an instrument one-eighth of an inch in diameter. The sinus was washed out and the contents removed, followed by injections of a 25 per cent solution of argyrol.

Subsequent daily applications of a similar character were made and in a week's time he was well. The argyrol had the happy effect of reducing the inflammation and checking hypersecretion of the sinus.

In conclusion, I beg to say that in the presentation of this report I do not wish to overestimate my observations nor appear too enthusiastic; simply to state what my experience has been and to declare that clinically, the antral theory of hay-fever and its allied disorder, asthma, has a basis for existence and recognition.

DISCUSSION.

DR. SOLENBERGER was of the opinion that many cases were reported as cases of hay-fever which in reality were not; he urged more care in diagnosis.

PAPER:

ON THE OBLITERATION OF THE ACCESSORY NASAL SINUSES.

A. R. SOLENBERGER, M. D.

I shall give a somewhat full report of this case, in order to set forth some points in the technique which have brought success in my hands. I wish the case also to serve as a type of conditions which clearly demand radical measures.

Judge B., age fifty-three, native of Ohio, asthma and bronchitis for seven years, consulted Dr. B. of New York during the fourth year of his disease; diagnosed bilateral ethmoiditis and nasal polypi. The latter were removed by the radical method of ethmoidal exenteration in the old way, by breaking down a few ethmoidal cells with the electro-motor but at intervals of several months,—a method originated by Dr. B. Thus at the end of six months the nasal polypi, together with their bases, were pronounced radically removed.

The patient returned to his home, to find a month later that instead of a diminution of nasal exudate, it had greatly increased.

Dr. H. of Buffalo was now consulted, who diagnosed *bilateral antral disease*. Both antra were now operated upon, through the canine fossae, curetted and treated by packing and irrigation, and the openings allowed to heal at the end of six weeks.

After the interval of some months the nasal discharge had in no way lessened, but rather *increased*, as well as the asthma and bronchitis. Moreover, there appeared a swelling over the *frontal sinuses*. Dr. H. curetted both frontal cavities through a single medial opening, and enlarged the naso-frontal ducts. The openings closed with great difficulty; indeed, in the patient's words, "I had *three* operations on the frontals under general anaesthesia, in *three months*."

There being still no cessation of the nasal discharge, the sphenoid cavities were investigated and found to discharge copious quantities of pus. These cavities were in turn operated upon, by enlargement of their openings and curettment.

The one feature of all these operative measures which seemed of especial significance to the *patient* was the secondary hemorrhage after the sphenoid work.

I saw the patient first a little over a year after this last operation. He had sought relief from his affliction in New Mexico and Colorado, hoping now that since all his nasal cavities had been scraped out, the climates, which had helped him before the operation, would now be even more beneficial. He thought his asthma and bronchitis were worse than ever. The quantity of pus from his head alone was over a pint daily. He now had for the first time since the operations again a painful swelling over the frontal sinuses. Meanwhile, too, the patient had been a diligent student of his own condition. He had heard that in Germany they were obliterating the nasal accessory cavities, and that Doctor C. in New York was then doing that operation on a relative of his. He wanted this done on all his cavities. He believed the Colorado climate would contribute to the success of the work. He would give all the time, and everything else that was necessary. He thought his physical condition equal to the radical work.

Of the six cavities, the frontals alone offered slight obstacles to canulization, due to the acute swelling. The patient was very desirous of being relieved of the painful acute frontal condition, to enable him to return east for a month to attend to a business matter involving a vast sum of money. This relief was speedily secured by enlarging the naso-frontal ducts from the inside, and irrigating the cavities. The irrigation was continued by the patient, and he was able to transact his business with a fair degree of comfort, and return to Colorado at the end of two months.

My study of the entire disease circle of this case during the time of treatment of the acute condition of the frontals convinced me that here was a case in which total cavity obliteration was in order.

The Frontal Sinuses. Finding the patient as enthusiastic as ever, we proceeded, under general anaesthesia, as follows: Incision through the median line of the brow, through its entire length, curving well down over the nasal process of the superior maxillary bone, and the removal of the entire anterior wall of the sinus. Every vestige of the overhanging wall was removed, so that the dips on all sides, if not slanting, were at least fairly vertical outward. The contours of both cavities were normal, that is, symmetrical in size, location and outline. Both were lined with pyogenic membrane, rath-

er loosely adherent, the entire cavity filled with suppurating granular tissue, easily spooned out *en masse*. The entire remnant walls were most thoroughly scraped, and irregularities leveled down as much as possible; most searching removal with curved currettes and burs of all necrotic tissue in the naso-frontal ducts, together with the destruction of one (remnant) ethmoidal cell, near the upper end of the duct. The cavities were then swabbed with carbolic acid and alcohol and packed down to, but not into, the nasal duct. No sutures were taken.

The first two weeks the repackings were done with very little pain, by local anaesthesia. At the end of that time there occurred the expected inveterate inversion of skin edges, in spite of vigorous under-packing—the beginnings of the dreaded deformities. The patient was anaesthetized and a thorough loosening and uplifting of the periosteum around the whole skin circle made a complete underpacking again possible. Meantime the cavities had half filled with a firm, healthy granulating tissue; the naso-frontal ducts were apparently filling in satisfactorily. After this, each week or ten days it became necessary to loosen again and lift up the skin edges, as they tended here and there to invert and prevent underpacking; this was now done under local anaesthesia, sometimes with the needle.

Thus by this periodical loosening and raising and underpacking of the inverting skin flaps, the naso-frontal ducts and the cavities filled up with good tissue, and the edges could be brought together with sutures at the end of the seventh week, and healing by first intention was complete by the eighth week. The flaps were fortunately so managed that when the hair appeared the two brows were on the same level. The only deformity noticeable was the slight depression above and between the nasal bones, occasioned by the previous operations. It is of interest here to note that, from the day of the operation on the frontal cavities, the patient had no more attacks of asthma.

The Sphenoid Cavities. The Sphenoid Cavities were operated on under local anaesthesia, in the sitting position. Cocaine and adrenalin were injected freely into the soft tissue around the enlarged openings, and afterwards powdered cocaine freely applied progressively. Although the openings were enlarged by previous operations, the cavities dipped down nearly two centimeters, which was about the depth of the remnant of the anterior walls. They were too hard and thick to be broken down by any hook or forceps in my posses-

sion. The only means at hand seemed to be the electro-motor bur. On the right side I began the work with some trepidation. This was the side where the secondary hemorrhage had occurred after the previous operation. I had meantime secured a reprint of the report which the former operator had given of this case. The hemorrhage had been alarming; the patient had had to suffer a retro-nasal packing for eight days, and was for a time in a state of collapse from exsanguination.

The operator advanced the opinion that in carrying his excavation toward the right he had exposed the spheno-palatine artery, a twig of the internal maxillary.

I, therefore, studiously tried to avoid drilling in that direction. I found that by removing a part of the partition between the two cavities; I could drill the anterior wall down to the floor of the cavity, and get a width of more than a quarter of an inch, through which I could make a very thorough curettment. I feared that by leaving a small part of the outer part of the anterior wall projecting, it would militate against obliteration; but in three weeks the cavity was fairly well filled with tissue, which in a few weeks more became quite firm. The other sinus was then proceeded with in the same manner. The first was filled out at the end of five weeks; the second, being somewhat smaller, filled out in four weeks. There was at no time any hemorrhage of any consequence. The patient had no asthma and only slight bronchitis during the three months of this operative work, and was otherwise in good physical condition.

The Maxillary Antrum. The Maxillary Antra were then opened, through the canine fossae, under general anaesthesia. The antra were found of such size that in order to secure working space to remove the entire anterior walls, especially to get a straight wall outward on the nasal side, the incisions underneath the lip had to be extended upward and inward close to the cartilaginous and osseous border of the nose; and, of course, outward well over the malar process of the maxilla.

Both cavities contained pyogenic membrane of various thickness, and granular tissue, especially on the floors and on the anterior walls apparently at the seat of former operations, and on the nasal wall, beginning at the hiatus, running downward to the floor and forward into the extreme anterior corner, large masses of tough pocketed tissue, all pus producing, was brought into view only after the removal of the extreme upper angle of the outer wall. I was satisfied that

this mass had not been removed in the former operation. This mass, as well as the pyogenic tissue around the hiatus, were too firm for the curette. It had to be removed with scissors and punch forceps.

The size of the antra, especially their antero-posterior diameter, being nearly two inches, made it seem a formidable proposition for obliteration, but a fairly good, funnel-shaped outward opening was secured, and a thorough curettment of the nasal openings to encourage their closure being made, the after treatment, aiming at a filling up of the cavity from the hiatus downward, was begun; i.e., by packing during the first week, loosely and at times afterward, firmly, to press downward too exuberant granular tissue, for three weeks; after which for a day or two, only now and then the packing was used. No irrigation was used, and the greatest care was taken in the dry cleansing and packing not to destroy the abundant delicate granulations. After the fourth week the dry cleansings were done only on alternate days.

As in the case of the frontal sinuses, the soft tissues around the openings had to be kept back by almost weekly incisions, and lifting of the periosteum (which, however, could always be comfortably done under local anaesthesia).

The opening into the nose in the one cavity closed pretty promptly, and this cavity filled out with much less attention than the other, whose hiatus did not obliterate but formed the inner end of a fistula, closing only in four months after numerous curettings of pus-producing tissue.

It should be said that during the entire time of this obliteration work, the nasal passages, whose linings, as might be supposed, were also at least in a semi-pyogenic state, were constantly under treatment. The winter following this work, the patient for the first time in seven years could remain at his eastern home. No asthma, very little bronchitis, and when last heard from, April 1st, 1906, two years after the last operative work, there was a very small quantity of non-purulent discharge from his nasal passages.

From my experience in obliteration work, I would advance a few considerations in reference to the indications for this radical work, and some points in the technique which I think are essentials to success.

First: In the case of any or all the cavities, the degree of chronicity should be determined as a guide in the choice of methods.

Second: The intra-nasal disease, which is practically always present, should be as nearly as possible eradicated. This in most cases being not possible, the treatment of the nasal passages should go on *pari passu* with the operative work, to guard against re-infection of the operative field.

Third: In all the cavities, too, the entire walls should be removed: no overhanging bones should be allowed; a funnel-shaped outward opening should be aimed at. Curettments should be religiously thorough; bare bone, even though not rough, should be vigorously scraped.

In the case of the frontal cavities, all the necrotic tissue should be scraped or burred out of the naso-frontal ducts, and in any event that canal should be so enlarged that any ethmoidal cells lying along

its side, or at the bottom, can be entirely obliterated. The packing should not enter into the duct. This cell obliteration, in several of my cases, left a duct large enough to admit the passage of the little finger into the nose and no trouble was experienced in the subsequent closure of the duct.

The form and location of the frontal cavities also should be taken into account when obliteration is thought of. This, indeed, I think should be the first consideration in the choice of any method of operation for the frontal cavities. Fortunately skiagraphy, in many cases, will show us the height and breadth of cavities, complete and incomplete septa, etc.

At most, only one suture should be taken at the outer angle, and one at the inner angle only when the incision has been carried far forward on to the nose.

I here show a device which is of considerable help in guarding against the inversion of the skin edges. This bridge is well retained and packing through its opening relieves much of the discomfort to the patient. It is made of malleable block tin.

Irrigation should be avoided as much as possible. The dry cleansings with swabs, at most only moist with peroxide if there is purulent matter, should be gently done, that the abundant delicate granulations be not bruised; but even more than in ordinary granulating wounds, one must know when to pack lightly or firmly or not at all, in order to secure the most rapid growth of tissue with plenty of the fibroid element.

Maxillary Sinuses: The Maxillary cavities offer special difficulties in their obliteration, mainly because of the distance of the nasal openings to the outer edges of the wound, and of the greater tendency to proliferation of the soft tissues around the external opening. Nature seems here to be especially rebellious against the obliterative aims of the surgeon.

The first condition for success here is the early closure of the hiatus semilunaris. To effect a closure of this opening is to work squarely against every law of nature which intends that this cavity should breathe through the nose and drain through the nose. Of course the same can be said of other sinuses, but when an attempt is made to heal the antrum through the canine fossae, a vast difference is experienced.

Other operators, too, have met with but indifferent success in obliterating these cavities. Among them Coakley, who has had perhaps the largest experience in this country, reports cases which still had fistulae a year after the operation.

For this reason I think obliteration of the maxillary cavities will in the future be more and more done by making vents and draining through nature's avenues; that is, by the Campbell-Luc method —the removal of the nasal wall and the encouragement of the filling up process. Indeed, by this method I have myself seen these cavities fill up with firm fibroid tissue, so that there was left only a shallow sulcus in the nasal fossae, and secretions that gathered there could be dislodged by ordinary blowing of the nose.

INDICATIONS FOR OBLITERATION OF THE SINUSES.

To in any way exhaustively study the indications for sinus obliteration, though perhaps the most profitble study of all, would not be

possible at this time. I would therefore consider the indications only in a general way.

First, in regard to all the cavities. As in all surgical work in which the method aims at the obliteration of a *structure,* not to say an *organ,* the *more radical* such *procedure* the *finer* should be the *discrimination* and the more deliberate the *judgment* in regard to the *indication.*

In general it may be said that the method is justified only in the highly chronic conditions, in multiple sinusitis; especially such as have had previous unsuccessful operations.

Second, in the case of the frontal cavities, the form and location should be taken into account when obliteration is thought of. This, indeed, should be the first consideration in the choice of any method of operation upon the frontal cavities. Fortunately skiagraphy, in many cases, will show us the height and breadth of the cavities, complete and incomplete septa. It will show certain forms of cavities that by *any method* which aims at *obliteration,* will cause such a deformity as to be considered by the *patient* a very extreme price to pay for his relief.

Third. Though we should be very tardy in discrediting a master in rhinological work, Sir Felix Semon, who recently, in a discussion of this subject, advocated extreme *conservatism* in *any radical* work upon the accessory cavities, giving for his reason that he has seen no cases *coming to grief,* we all know that we are derelict in our work if we do not act in the radical way, to permanently relieve certain conditions, which if they do not threaten life at least make life not worth living. Obviously it depends upon what constitutes the condition which Sir Felix calls "grief." Usually in all cases of multiple sinusitis, or even in many cases of chronic *frontal* sinusitis, there is a mental state present which ranges all the way from a depression which incapacitates the patient to states bordering upon suicide.

Aside from these serious conditions, we must all agree that reservoirs of pus whose geographical position are such as to constitute a prolific causal factor in the production of the numerous respiratory diseases make the obliteration not only desirable but obligatory. How frequently do we not find disease of the lungs only a segment in the disease circle running through the cavities of the head which are in a state of chronic suppuration? How futile and often impossible is the application of remedial measures to diseased cavities in

the lower respiratory tract while chronic inflammatory and often infectious exudates from the upper cavities drain down upon them!

In this respect it will be plainly seen that the frontal cavities are the greater sinners; and as a matter of fact, of all the accessory sinuses the obliteration of the latter is the most frequently indicated. The fact that the maxillary sinuses are the most frequently found diseased is no doubt due to their location relative to the frontal sinuses and anterior ethmoid cells—their funnel-shaped openings invite the dumpings from these upper cavities and convert them into receptacles for these dumpings.

In regard to the destruction of any function that these cavities may have, it may be perhaps sufficient to say that we must choose between the least of two evils.

Fourth. After ruling out elderly people and people who have other serious diseases, as of the heart, kidney and lungs, we may, I think, say that all other cases which have had effective intra-nasal treatment, without cure, if they desire to be permanently rid of their diseased cavities, must submit to the obliterative work.

In answer to a query recently put to Coakley, he says:

"I firmly believe that obliteration of the accessory sinuses is the only sure and permanent cure for chronic suppurative sinusitis. The method by which this is to be accomplished must vary considerably with individual cases. While I have been exceedingly successful with the methods advocated in the aforesaid reprint,* wider experience has led me to believe that in some cases other methods of treatment accomplish the same result with less discomfort to the patient."

I think we can all agree with Dr. Coakley's general statement; but in it we no doubt all see an opportunity and the necessity for fine discrimination as to just what class of cases comes under the head of the "some cases" referred to in his modifying statement. I would more especially make it apply to those of the maxillary sinuses—in which other methods "will produce the same results with less discomfort to the patient."

Fifth. There are numerous other considerations, for the most part such as affect the patient.

In general, the whole patient must enter into the problem. The *man* must be considered. It is obvious that he must be rather extraordinarily qualified, at least in three respects: 1st: Morally, he must have more than ordinary patience and faith in the operator.

2nd: Intellectually, he should have a more than ordinary understanding of the condition, procedure and result expected in order to insure that patience. 3rd: Physically more than ordinary endurance. And a 4th qualification might be added, financially. As human nature and conditions are constituted, this may make for or against the success.

Sixth. Again, it is not always possible to take in every factor that will determine the method of procedure until the cavities are actually opened and explored.

Not to prolong this presentation, I would refer the fellows to Dr. Coakley's exhaustive resumé, for further conditions that call for the open operative method, in his admirable paper on "Frontal Sinusitis; Diagnosis and Treatment."

DISCUSSION.

DR. GALLAHER laid stress on the anatomic relationship of the Bulla Ethmoidalis, Hiatus Semilunaris and the Uncinate Process of the Middle Turbinal; a deviation of the normal anatomical relationship being responsible for many ocular cases of apparently obscure etiology. Abscence of pus was not, in itself, contradictory to such etiology.

PAPER:

TOOTH CYSTS.

THOS. J. GALLAHER, A. M., M. D.

It is not our purpose to transgress upon the domain of the dentist, but rather to report a few cases of interest to the rhinologist.

Tooth cysts may be divided into periosteal and follicular cysts. Periosteal cysts have their origin in the covering of the root, and consist of granulation and epithelial cells. They appear to be more frequent in the upper jaw than the lower, although they are many times overlooked in the lower jaw, owing to the thickness of the bony cortex limiting their growth.

It has been my experience that the lateral incisor and bicuspid are especially affected. The sack gradually increases in size, encroaching upon the surrounding parts, and many times, owing to

the bony covering being absorbed, extends into the antrum and nose. It contains a straw-colored fluid, often rich in cholesterin crystals. Not infrequently the contents are infected and a pus sack results.

The treatment of the periosteal cyst is simple and efficient. Incision is made into the cyst wall, thoroughly draining it. If necessary, a counter-opening is made and very mild packing is used for several days, after which time washing of the cyst with antiseptic solutions is all that is required. A cure is usually obtained in from three to six weeks.

The Follicular cysts originate in normal or supernumerary retained tooth germs. They are much slower in growth and not at all dependent upon pathological changes in the tooth itself. The diagnosis is sometimes difficult, but with a swelling in either jaw, together with the absence of the permanent tooth, a Dentigerous cyst should be suspected. The use of the x-ray photograph is especially useful for diagnostic purposes.

The Follicular cysts are the true Dentigerous cysts — that is, tooth-bearing cysts. A root is never developed from these germs, but a tooth crown is often found in the sack.

Treatment of Follicular Cysts.—An incision is made through the mucous membrane and periosteum and a large flap is dissected loose. After chiseling away the bony cortex, the inner cystic wall should be most thoroughly dissected out and the mucous membrane of the periosteum crowded into the cavity by a compress. If the sack is not thoroughly removed, there may be a recurrence. Not the least of the dangers of a dentigerous cyst is the possible occurrence of a rarifying osteitis which may destroy the entire jaw.

Case I.—Miss R., age 25, presents herself with marked facial deformity. There is a swelling underneath the upper lip, on each side of the median line, so prominent that the nose is displaced forward. Upon examination, a cyst on each side is easily detected, at the junction of the alveolar and buccal mucous membrane. Examining the nose internally, a cyst is seen along its floor and underneath the anterior portion of the inferior turbinate body. This is a very plain case of double Periosteal cyst connected with the right and left incisor. This is the first case of double Periosteal cyst that I have ever seen. Two incisions were made into the sack, one in the nose and one in the mouth, giving good drainage. This was followed by gentle packing, stimulation with a mild so-

lution of iodin and washing. Both cysts were cured within five weeks. After the operation the cosmetic effect was very striking.

Case II. Mrs. R., age 35, presents herself with a swelling at the right of the median line, causing a displacement of the wing of the nose. It has been coming on for some months and has been painful during the last week. Intranasal examination shows a swelling on the floor of the nose extending to the outer wall. By direct palpation in the nose and under the lip, a cyst is diagnosed. An incision under the lip liberated pus. Another incision was also made on the floor of the nose. The usual treatment was followed out, resulting in a prompt cure.

Case III. Exactly like *Case I,* occurring in a woman, age 40. The sac contained pus and was unilateral.

The above are all illustrations of the ordinary Periosteal cysts.

In conclusion, I wish to report two cases of Follicular or Dentigerous cysts.

Case I. Boy, age 11, presents himself with a swelling in the upper left maxilla which is firm and painful. Immediately above the sixth-year molar a Dentigerous cyst was suspected, and we operated upon him a few days afterward. With a good labial retractor, the parts were well exposed and incision made from the canine fossa posteriorly to the malar ridge. The membrane and the periosteum were carefully dissected loose, followed by two incisions at right angles to the original one, and the bony cortex removed. The cyst was then dissected out as thoroughly as possible, followed by curettage. The membrane, with its periosteum, was then pushed back into the cavity and held in position by means of tampon. Suppuration followed, but after eight weeks of careful washing and stimulation the entire cavity closed. In this cyst an inverted tooth crown was found, probably a supernumerary bicuspid.

Case II. Young woman, age 22. A diffuse hard swelling is found over the second bicuspid. She reports that she was operated upon previously and the bone scraped. There is a large fistula opening into the antrum. This fistula has discharged for over a year. There has been no discharge of pus from the nose. An incision was made from the median line to the malar ridge. The membrane was then carefully dissected up. In this case it became necessary to remove the entire anterior wall of the antrum. The cyst wall was removed and the extensive removal of bone

was made necessary by the existing osteitis. The walls of the antrum were also curetted. The subsequent treatment consisted in the packing and washing of the antrum. A drainage tube was soon introduced and the washing continued through it. Healing was complete in about seven weeks.

No doubt every rhinologist sees many of these cases, but as all of these had been overlooked, I hope that the recital of my experience will be of some interest to you.

PAPER:

A TREATMENT OF ACUTE OTITIS MEDIA.

P. F. GILDEA, M.D.

During the past six or seven years I have had marked success in the treatment of acute otitis, by the following method:

When inspection shows any redness of the drum, with or without pain, I paint it with a 10 per cent solution of Ichthyol, once or twice a day. This can be done without pain, if the cotton is allowed to project one-eighth inch beyond the applicator. The solution may be either cold or hot, and it will often stop pain in a few minutes. Then I order 10 to 20 drops of this solution to be dropped warm in the ear every hour until relieved.

When the nose is blocked, I cocainize the inferior turbinte on the affected side, and make a short line with pure trichloracetic acid on it and immediately neutralize the acid with carbolated vaseline, after which I paint the Eustachian cushion with 5 per cent Ichthyol and immediately wash off with a weak Dobell's Solution spray.

I have seen bulging membranes reduced without rupture, and pus inside the drum, disappear without operation in twelve hours. I have never had mastoiditis where I could carry out this plan. Trusting that some of you may be equally successful with the method, I submit it to you for consideration.

DISCUSSION.

DR. LEVY, of Denver, has employed ichthyol for some time; he uses a solution in glycerine and after painting the membrane leaves a wick of gauze saturated with the solution in the external canal; he attributes the results obtained largely to the glycerine and does not think the treatment will either abort cases or prevent pus.

PAPER:

OCULAR DISEASES OF NASAL ORIGIN.

JAMES ALLEN PATTERSON, M. D.

The immense strides made in Ophthalmology and Rhinology in recent years, particularly rapid in Rhinology, has kept the independent worker in these specialties so busy apparently that the interdependence of nasal and ocular maladies has been partially lost sight of. Even now, as well known as is the relationship of lachrymal diseases to nasal disorders, we find nasal treatment largely neglected.

As a student in Ophthalmology, I observed that a metal canula introduced into the sac and down the duct into the nose through which boracic solution was syringed, gave in many instances better results than simply probing the duct. It was surely a painful method of nasal irrigation.

I am not ready with sufficient data to prove an assertion that the majority of cases of lachrymal abscesses are of ethmoid origin; but the fact that in so many of these cases an attempt to probe the duct will disclose roughened bone and that unless great care and skill are exercised the probe will enter the nostrils behind the duct through the ethmoid cells rather than through the lachrymal passage makes my belief tenable. I do not assert that we do not get some catarrhal conditions carried from the eye downward, but I am strongly of the belief that conditions of this sort are in the minority.

It has been my habit where a conjunctivitis is complained of and the conjunctival congestion is largely in the lower lids rather than the upper or the bulbar conjunctivae, to suspect the lachrymal passages and still further the nose of being the focus. Only a few weeks ago I had a striking example. I was consulted by a lad of 14 years, who came for conjunctivitis of the left eye only. There was hyperæmia of the skin of both the upper and lower lids, the patient being of a florid complexion made such more noticeable. The

unilateral conditions led me to suspect a foreign body, as the complaint was of only three or four days' duration. Search for such was negative. I was, however, impressed by the fact that the congestion of the conjunctivæ was confined almost exclusively to the lower bulbar and lower palpebral conjunctivæ. Further questioning elicited the fact that there was pain of this number of days standing limited to the left brow, temple and less severely the malar eminence. A further history of rhinitis of a week's duration was obtained. Examination of the nose showed bogginess of the middle turbinate, and after cocain and adrenalin dilatation, pus was seen coming from the neighborhood of the left ostium naturale of the antrum. Drainage and irrigation soon cured pain, conjunctivitis and catarrhal conditions. The patient had enlarged tonsils and adenoids which were subsequently treated surgically.

Vieusse[1] and others believe that tuberculosis of the conjunctivæ sometimes arises from extension of the unrecognized nasal tuberculosis.

I think we are only in the infancy of our knowledge of the causative relationship of the nose to some forms of keratitis. At least, those of us who have had our hard knocks treating phlyctenular keratitis in the young are impressed with the fact that we had better not neglect the nostrils unless we take the chances of having our reputations injured. I have had, as you no doubt have had, the most brilliant cures in this malady following an adenotomy with or without tonsillotomy, and I believe I cannot urge too strongly the necessity of thorough nasal exploration in all forms of recurring keratitis.

A few days ago I did an iridectomy for optical purposes on the left eye of a boy of 16 years, who had very large tonsils. It was necessary to give a general anasthetic. Permission to remove tonsils a few weeks before the operation on the eye was refused. Anaesthesia was induced only after a long preliminary struggle. Twenty-four hours after the operation five punctate superficial ulcers appeared upon the unoperated eye and subsided in twelve hours under argyrol and iodoform ointment. Forty-eight hours afterward the disease reappeared in the same eye, although the distribution of the areas was different, this subsided in twelve hours under the same treatment and did not recur when daily spraying of the nostrils with a simple saline solution was instituted. The nose had

been treated daily for a week previous to the operation in order to prevent any complications.

I believe the case reported by Schneideman[2] in 1904, of Central Superficial Choroiditis of one eye only, to have been due to pent up sphenoidal secretions discovered when pus was found to be flowing from that sinus; the choroiditis as reported rapidly improving from that time. It is a most difficult thing to exclude as a non-focal point a sphenoidal sinus which is not discharging, and in many such instances not to be reached by a probe without previous surgical removal of a middle turbinate.

Schmieglow[3] has recently reported "two cases of Retro-bulbar Optic Neuritis due to Sphenoidal and Ethmoidal Sinusitis."

The literature of asthenopic symptoms, contracted fields, affections of extra ocular muscles, pupillary changes, etc., have been gone over so well by Posey[4] that it is useless to repeat them.

The pathology I believe to be one of absorption from diseased conditions in the superior and middle meatus of the nose, the anatomical reasons for which I have previously set forth in an article [5]"Concerning the relationship of Nasal Disorders to Vitreous Opacities." I wish I could have illustrated that article with the excellent cuts of the "Arterial connection between the nose and its accessory sinus," and the illustration from Zuckerkandl which C. R. Holmes[6] has made use of in a monograph, "Head Pains and Eye Symptoms caused by Inflammation of the Accessory Sinuses of the Nose."

Dr. Holmes has well said that "lowered blood pressure and slower circulation (during sleep) * * * cause the secretions of the nose to flow more slowly, and favor inspissation, and that decomposition is constantly going on." It is really wonderful how well the eye is protected from the diseases that immediately surround it. Its bony envelope, its elevation above the nasal cesspool and its arterial supply from the deep carotid rather than from superficial trunks make it a veritable Port Arthur. Yet, like the Russian stronghold, prolonged siege will make inroads. My previous observations above noted have shown that when there is good drainage from any of the accessory sinuses the deeper tissues of the eye are comparatively free from attack, but even a small focus of pent up secretions and their decomposition will make inroads upon its integrity, either by erosion of its bony envelope, extension by contiguity or absorption, as previously stated. Ziems theory of "passive

orbital venous Stasis," as pointed out by Manning Fish[7] in a very large and most interesting series of cases of "Uveitis due to Accessory Sinus Diseases," is admitted, but this must account for cases showing the more acute symptoms of pain, more or less severe, of rapid rather than sluggish onset, together with the accompanying symptons, such as accommodative disturbance. Its continuance favors absorbtion of peccant material.

In all cases of unilateral ocular maladies the nose should never be forgotten as possibly being able to furnish evidences of the cause of the disease.

Strader of Cheyenne[8] has recorded a case of glaucoma markedly benefited by treatment of the nostril of the corresponding side by suction methods. I have verified his findings in relieving cases of acute sinusitis by this method. Frank E. Brawley has devised an ingenious instrument for the purpose.

I desire to report the following cases:

Case 1. This case is briefly reported to the Colorado Ophthalmological Society.[9] J. K., age 39, a railroad section boss, was first seen in June, 1905. He had entered a general hospital some few weeks before, where he was said to have been treated for severe neuralgia of the head. The pain being most intense in the right temple and so severe as to prevent sleep. Eventually proptosis 3mm. and diplopia occurred, when Dr. E. R. Neeper saw him, who found at that time T+1. As there was a suspicion of their being a nasal complication, I looked over the case with Dr. Neeper.

At this time, June 23, O. D.v 5/10, O. S.v 5/5. Tension had fallen to normal under pilocarpine collyria prescribed by Dr. Neeper. There was a mild papillitis of the nerve of O. D. with fine flame-shaped exudation radiating from the nerve up, in and down, the media was, possibly, slightly hazy. The eye ground of O. S. was in health. He is unable to move O. D. out beyond the middle line and the movement in was possibly slightly restricted, he had full movement downward, but the ability to turn the eye upward was materially interfered with. Proptosis from my scale, 8mm.

Under cocaine I curetted the ethmoids seemingly as far back as they extended, the tip of the curette entered 9mm. from the floor of the vestibule; both frontal sinuses transillumined clear. It was impossible to probe the right owing to a deflected septum.

Pain speedily lessened after this drainage and the proptosis gradually subsided. Five days afterward vision was 5/5, the papillitis was subsiding and the media much clearer; diplopia homonymous with R. H. 6°. July 15, my notes give v=5/5, papillitis had sub-

sided and proptosis not noticeable. Diplopia complained of only on looking to his extreme right.

Several curettements were made of the ethmoids and when last seen, on August 15, the v=5/4, eye grounds were normal, diplopia only on looking to his extreme right. The patient returned to work though there was still pus flowing from the ethmoid cells. Consent to further operative proceedure was denied.

Case 11. J. J. C., male, age 38, was first seen on April 28, 1904, presenting oedema, and redness of the skin above the inner canthus of the right eye. The case had been treated as erysipelas for some days. This abscess was incised and pus liberated. The case did not return until the following July 16, when there was found a sinus at the point of my incision through which a large amount of pus had discharged daily. Pus was found in the middle nasal meatus which was occluded by a polypus. Transillumination showed a faint shadow in the lower part of the right frontal sinus with light transillumination of the right maxillary antrum a trifle less than that of the left. O. D. was then in health. Operation was advised but consent to it not obtained.

This case was not seen again until December 17, 1906. The sinus previously described remains although the amount of pus coming from it was much less. A probe enters 25mm. by carrying it backward and upward at an agle of 30°. Middle turbinal is enlarged and polypoid. No pus could be obtained from the frontal duct.

He gives a history of great pain in O. D. for the past year, sufficient at many times to keep him awake at night. During this time, the sight in this eye has declined to light perception only. The cornea is now uniformly opaque nothing, not even the iris, can be seen through it; there are some fine vessels upon its surface; the conjunctiva is white; T. N.; vision in his left eye is 5/3. A pathetic evidence of neglect.

Case 3. A lad of 14 years consulted me a week ago for pain in the left eye, the entire frontal region and left temple. This was accompanied by intense photophobia of the left eye with some lachrymation. He was unable to attend school for the past two days owing to the discomfort and inability to use his eyes. No foreign body could be found nor any form of corneal disease, the lower conjunctival cul de sac was distinctly conjested the right to a very much less degree; there was no pus in the eye. The lids had agglutinated the night before. He has worn satisfactorily and comfortably for five months the astigmatic correction +0.75 cy., ax. 90, in each eye, which gave him normal vision, the eye grounds were in health. History of a mild rhinitis of three or four days duration was learned.

In the left nostril, after prolonged exploring, muco-pus was liberated from the anterior ethmoid cells. Pain in his eye ceased while he was in my operating chair, the headache continued and was not relieved by suction. The next day muco-pus was flowing unobstructedly in limited amount, coming down between the middle turbinate and the outer wall. All pains complained of had ceased, photophobia and lachrymation had disappeared.

CONCLUSION.

You will remember that some years ago the progress of otology was hindered by not having the nasal source of its many diseases treated as well as now. I believe that ophthalmology, while far less dependent upon rhinology, can learn much to its advantage by the interdependence of the two special practices.

BIBLIOGRAPHY.

1. Quoted by Stevens in *Colorado Medicine*, January, 1907.
2. Transactions Amer. Academy Ophth. and Oto. Laryngology, 1904.
3. Schmiegelow, E.—"Hospitalstidende." See *Journ. A. M. A.*, Vol.
4. Posey, W. C.—Trans. Ophth. Section A. M. A., 1905.
5. Patterson, J. A.—*Ophthal. Record*, March, 1905.
6. Holmes, C. R.—*Ohio Med. Journ.*, February, 1906.
7. Fish, Manning—*Amer. Journ. of Ophthal.*, December, 1904.
8. S rader—*Oph. Record*, Trans. Colo. Oph. Society, January, 1907.
9. Patterson, J. A.—*Ophth. Record*, Vol. XIV., page 591.

IN MEMORIAM.

SAMUEL EDWIN SOLLY.

At the last meeting of the American Laryngological, Rhinological and Otological Society—held at Kansas City—it was made known to the members of this organization that our once honored President and much-loved fellow was seriously visited by illness that had no prospect but a fatal termination. During his illness, Dr. Solly visited various places in order to obtain relief from his physical suffering, at last finding that solace by being taken into the arms of his Creator at Asheville, on November 18, 1906. Dr. Solly was one of the early members of this organization and its fourth President. It is almost impossible to write an obituary of this remarkable man without appearing fulsome. He prized most highly his membership in this society and rarely failed to attend our annual meeting, where his genial countenance, his kindly spirit, his helpful word, his courteous manner, his bright and witty remarks and his learned papers and well-considered discussions endeared him to all. He was a man of remarkable attainments—a representative of the highest type of the intellectual Englishman; in medicine, he was a scholar with an investigative turn of mind; an executive of remarkable ability; a public-spirited citizen of the highest grade; and a writer upon climatological subjects whose fame and name are co-extensive with the realms of modern medicine. His personality was the most delightful that the writer of this article has ever known. He possessed a quiet and dignified bearing, a modest temperament, a genial disposition, kindness of thought and action, a courtesy of manner that never was at fault, a ready wit that never lent itself to sarcasm, a perfect host, a most welcome guest, strong intellectually, with high moral ideals, and the courage of his convictions—such was the character of the lovable man of whom we write and who won his way into the hearts of all who came within his presence.

Dr. Solly was born in London, England, May 5, 1845. He received his early education in the best schools of his native land, and, early following the footsteps of his distinguished father, he took up a medical career. Owing to a physical breakdown in his early career, his attention was naturally directed along climatological lines. Through his father, he was brought into intimate association with many of the distinguished practitioners of London. In this way, he became acquainted with the late Sir Morell Mackenzie, from whom he acquired his insight into laryngology and rhinology. In his thirtieth year, he found it impracticable, from a physical point of view, to longer reside in London, so he turned his face towards the setting sun. We next find him in Manitou, where he resided for some years, being the inspiration of the English colony at this place. While at Manitou he made an investigation of the mineral springs of that place. Shortly after the establishment of the city of Colorado Springs, he removed to this more inviting and thriving town. His great life work was done at Colorado Springs. He never tired of singing its praises or writing of its glories. Several years ago he was very much interested in a movement started by the civic body of Colorado Springs in sending out the secretary of that organization on a tour of the principal cities of the country to proclaim the attractions of Colorado Springs as a sanitarium. Though much interested in all projects of a civic character that redounded to the benefit of his home city, he was much more interested in those projects which had for its purpose the recognition of the fact that it was the world's great sanitarium for those afflicted with tubercular affection. The Cragmoor Sanitarium was his ruling passion during the last few years of his life. This institution was modeled on a most comprehensive system, as only the master hand of Solly could have formulated.

The following autobiography was furnished me by Dr. P. F. Gildea, his associate, who states that it came from the hands of Dr. Solly. C. W. R.

WILLIAM PARTRIDGE BRANDEGEE.

William Partridge Brandegee was born in Brooklyn, N. Y., April 16, 1864. He was the son of Marius Brandegee, who was a graduate of Yale University in 1843, and of Catherine Amelia Fountain.

He attended the Pingry Preparatory School of Elizabeth, N. J., from boyhood, until his entrance at Yale in 1882, from whence he was graduated from the Academic Department in the class of 1886. The following autumn he matriculated at the College of Physicians and Surgeons, the Medical Department of Columbia University, from whence he was graduated in 1889.

Immediately following his medical graduation he took up the special practice of nose, throat and ear diseases at the Manhattan Eye and Ear Hospital, and served as an assistant surgeon in the Throat Department of that institution from 1890 to 1897. He was connected with the Aural Department of the New York Eye and Ear Infirmary as assistant surgeon from 1896 to the time of his death, July, 1907. In 1899 he became chief of clinic in the Aural Department of the University and Bellevue Hospital Medical College, which position he held until his death last July.

Dr. Brandegee was a member of the American Laryngological, Rhinological and Otological Society, the American Otological Society, the New York Otological Society, the Medical Society of the County and State of New York, and the Medical Society of Greater New York. From 1899 until the fall of 1906 he was associated in practice with Dr. Edward B. Dench. At the time of his death he was actively engaged in the practice of our specialty at 40 West Forty-eighth street, New York City.

He was married to Caroline Morris in 1888, who, with one daughter and two sons, survives him.

By the death of Dr. Brandegee, this Society has lost one of its most enthusiastic and earnest workers, and those of us who were privileged to know him intimately, a true friend, whose every thought and action, both in his professional and private life, were imbued with the highest sense of honor. J. F. M.

BUSINESS MEETING.

The thirteenth annual meeting of the American Laryngological, Rhinological and Otological Society was held at the New York Academy of Medicine, New York City, on Thursday, May 30, 1907, at 10 o'clock. During the sessions of the Society the following Fellows registered:

Jos. H. Abraham, New York.
John L. Adams, New York.
Willis S. Anderson, Detroit, Mich.
F. C. Ard, Plainfield, N. J.
Hubert Arrowsmith, Brooklyn, N. Y.
W. L. Ballenger, Chicago, Ill
John F. Barnhill, Indianapolis, Ind.
Clarence J. Blake, Boston, Mass.
Jos. Beck, Chicago, Ill.
Charles B. C. Borden, Boston, Mass.
J. Price Brown, Toronto, Canada.
W. S. Bryant, New York.
Bishop R. Canfield, Ann Arbor, Mich.
T. R. Chambers, Jersey City, N. J.
W. E. Chenery, Boston, Mass.
W. A. Chisholm, New York.
C. G. Coakley, New York.
George M. Coates, Philadelphia, Pa.
F. C. Cobb, Boston, Mass.
L. A. Coffin, New York.
Henry Coggeshall, New York.
J. Solis Cohen, Philadelphia, Pa.
T. Corwin, Newark, N. J.
A. Coolidge, Boston, Mass.
G. F. Cott, Buffalo, N. Y.
C. N. Cox, Brooklyn, N. Y.
R. H. Craig, Montreal, Canada.

Frank M. Cunningham, Macon, Ga.
W. L. Culbert, New York.
J. F. Culp, Harrisburg, Pa.
H. H. Curtis, New York.
Ewing W. Day, Pittsburg, Pa.
W. S. Daly, Ogdensburg, N. Y.
Edward B. Dench, New York.
Arthur B. Duel, New York.
H. S. Dye, Washington, D. C.
W. P. Eagleton, Newark, N. J.
Bert H. Ellis, Los Angeles, Cal.
Linn Emerson, Orange, N. J.
F. P. Emerson, Boston, Mass.
T. H. Farrell, Utica, N. Y.
E. W. Fleming, Los Angeles, Cal.
John M. Foster, Denver, Colo.
T. J. Gallaher, Denver, Colo.
P. G. Goldsmith, Belleville, Ont.
M. A. Goldstein, St. Louis, Mo.
Charles Graef, New York.
C. P. Grayson, Philadelphia, Pa.
Thomas H. Halsted, Syracuse, N. Y.
W. G. B., Harland, Philadelphia, Pa.
H. J. Hartz, Detroit, Mich.
W. H. Haskin, New York.
N. J. Hepburn, New York.
F. W. Hinkel, Buffalo, N. Y.
C. R. Holmes, Cincinnati, Ohio.
E. M. Holmes, Boston, Mass.
D. L. Hubbard, New York.
Thomas Hubbard, Toledo, Ohio.
Lee M. Hurd, New York.
Thomas J. Harris, New York.
R. Johnson Held, New York.
E. F. Ingals, Chicago, Ill.
J. M. Ingersoll, Cleveland, Ohio.
F. L. Jack, Boston, Mass.
Chevalier Jackson, Pittsburg, Pa.
C. P. Jones, Newport News, Va.
G. F. Keiper, Lafayette, Ind.

S. J. Kopetzky, New York.
B. R. Kennon, Norfolk, Va.
J. L. Kent, Lynchburg, Va.
I. E. Kimball, Portland, Me.
F. H. Koyle, Hornellsville, N. Y.
D. A. Kuyk, Richmond, Va.
D. B. Kyle, Philadelphia, Pa.
J. J. Kyle, Indianapolis, Ind.
M. D. Lederman, New York.
R. Levy, Denver, Colo.
R. Lewis, Jr., New York.
H. W. Loeb, St. Louis, Mo.
J. E. Logan, Kansas City, Mo.
S. H. Lutz, Brooklyn, N. Y.
G. Hudson-Makuen, Philadelphia, Pa.
C. A. Manson, New York.
W. B. Mason, Washington, D. C.
L. L. Mial, New York.
L. H. Miller, Brooklyn, N. Y.
F. E. Miller, New York.
T. W. Moore, Huntington, W. Va.
H. P. Mosher, Boston, Mass.
C. E. Munger, Waterbury, Conn.
G. D. Murray, Scranton, Pa.
H. L. Myers, Norfolk, Va.
Robert C. Myles, New York.
James F. M'Caw, Watertown, N. Y.
S. McCullagh, New York.
John Wesley Murphy, Cincinnati, Ohio.
L. A. McClelland, Brooklyn, N. Y.
J. McCoy, New York.
J. F. McKernon, New York.
O. A. M. McKimmie, Washington, D. C.
F. H. McLaury, New York.
J. O. McReynolds, Dallas, Tex.
H. J. Off, Philadelphia, Pa.
S. Oppenheimer, New York.
W. C. Phillips, New York.
J. B. Rae, New York.
G. L. Richards, Fall River, Mass.

Henry O. Reik, Baltimore, Md.
J. D. Richards, New York.
C. D. Richardson, Washington, D. C.
W. H. Roberts, Pasadena, Cal.
G. T. Ross, Montreal, Canada.
Dunbar Roy, Atlanta, Ga.
C. C. Sandles, Pittsburg, Pa.
J. E. Sheppard, Brooklyn, N. Y.
J. H. Shorter, Macon, Ga.
S. MacCuen Smith, Philadelphia, Pa.
E. T. Smith, Hartford, Conn.
Herbert E. Smyth, Bridgeport, Conn.
S. F. Snow, Syracuse, N. Y.
Amos R. Solenberger, Colorado Springs, Colo.
Paul F. Sondern, Rochester, N. Y.
B. R. Shurly, Detroit, Mich.
W. E. Sauer, St. Louis, Mo.
F. B. Sprague, Providence, R. I.
O. J. Stein, Chicago, Ill.
G. C. Stout, Philadelphia, Pa.
J. A. Stucky, Lexington, Ky.
C. F. Theisin, Albany, N. Y.
F. M. Thigpen, Pensacola, Fla.
H. H. Tyson, New York.
J. J. Thomson, New York.
E. L. Vansant, Philadelphia, Pa.
A. P. Voislawsky, New York.
G. A. Webster, Boston, Mass.
W. A. Wells, Washington, D. C.
Ernest De Wolfe Wales, Boston, Mass.
N. L. Wilson, Elizabeth, N. J.
D. J. Gibb Wishart, Toronto, Canada.
D. G. Yates, New York.

Dr. John A. Wyeth, of New York City, President of the New York Academy of Medicine, welcomed the society on behalf of the members of the Academy, and Dr. J. A. Stucky, of Lexington, Ky., responded to the greeting.

The President, Dr. Wendell C. Phillips, of New York City, delivered the annual address.

The meeting was then formally declared open. Upon motion, seconded and carried, visiting members of the profession were invited to take part in the discussions.

Reports were received from the four Sections of the society, the Eastern, Middle, Western and Southern.

The scientific program which followed consisted in the presentation of cases, instruments and apparatus, and the reading of papers, with the discussion thereof.

At 3:15 p. m., the society met again and went into executive session, the President presiding.

The Secretary reported that the Council recommended for election as Fellows the following:

Clarence John Blake, M. D., Boston, Mass., proposed by Wendell C. Phillips, M. D., seconded by Eugene A. Crockett, M. D.

George Morrison Coates, M. D., Philadelphia, Pa., proposed by D. B. Kyle, M. D., seconded by Francis R. Packard, M. D.

Francis Patten Emerson, M. D., Boston, Mass., proposed by Eugene A. Crockett, M. D., seconded by John F. Barnhill, M. D.

John M. Foster, M. D., Denver, Colo., proposed by W. C. Bane, M. D., seconded by Robert Levy, M. D.

Samuel J. Kopetzky, M. D., New York, proposed by Wendell C. Phillips, M. D., seconded by Edward B. Dench, M. D.

Samuel McCullagh, M. D., New York, proposed by T. Passmore Berens, M. D., seconded by Wendell C. Phillips, M. D.

D. MacPherson, M. D., New York, proposed by Thomas J. Harris, M. D., seconded by Wendell C. Phillips, M. D.

John Wesley Murphy, M. D., Cincinnati, Ohio, proposed by Christian R. Holmes, M. D., seconded by J. A. Stucky, M. D.

John A. Robinson, M. D., New York, proposed by Edward B. Dench, M. D., seconded by James F. McKernon, M. D.

B. R. Shurly, M. D., Detroit, Mich., proposed by Henry J. Hartz, M. D., seconded by Thomas Hubbard, M. D.

William E. Sauer, M. D., St. Louis, Mo., proposed by Max A. Goldstein, M. D., seconded by W. B. Shields, M. D.

John J. Thomson, M. D., New York, proposed by Wendell C. Phillips, M. D., seconded by Edward B. Dench, M. D.

Wade Thrasher, M. D., Cincinnati, Ohio, proposed by John F. Barnhill, M. D., seconded by A. B. Thrasher, M. D.

Ernest De Wolfe Wales, M. D., Indianapolis, Ind., proposed by George A. Webster, M. D., seconded by William E. Chenery, M. D.

On motion, the Secretary was instructed to cast an affirmative ballot for the list of candidates as read, and they were declared duly elected.

Dr. Otto J. Stein, of Chicago, Ill., and Dr. Lee M. Hurd, of New York City, were appointed an auditing committee to audit the Treasurer's accounts.

The Treasurer's report was read and referred to the Auditing Committee.

The President appointed the following as the Nominating Committee: Dr. James F. McKernon, New York City; Dr. Chevalier Jackson, Pittsburg, Pa.; Dr. J. A. Stucky, Lexington, Ky.; Dr. James E. Logan, Kansas City, Mo., and Dr. Charles W. Richardson, Washington, D. C.

It was moved and seconded, and carried, that the President's address be referred to a committee for the consideration of the recommendations made therein, said committee to report at some session of this meeting. The following committee was appointed: Dr. E. Fletcher Ingalls, Chicago, Ill.; Dr. Frank B. Sprague, Providence, R. I.; Dr. D. Braden Kyle, Philadelphia; Dr. James E. Logan, Kansas City, Mo., and Dr. Christian R. Holmes, Cincinnati, Ohio.

The business meeting adjourned, and the society proceeded with the scientific program.

The Society went into executive session at 10:15 a. m. on Friday, May 31, the President presiding.

The Secretary presented his annual report—

Report of the Secretary.

The Secretary regrets to report the death during the past year of two members:

Dr. S. E. Solly, of Colorado Springs, Colo., November, 1906, and Dr. Wm. P. Brandegee, of New York, July 30th, 1906.

Following precedent, a copy of the Transactions of the Twelfth Annual Meeting is herewith presented. Many unavoidable delays have prevented its appearance till the present month, but, by this time, it should be in the hands of all the members. It is a volume of nearly 700 pages, the largest in our history, and is one that does credit in every respect to the Publication Committee. In this connection, it may be stated that the surplus volumes of the Transactions have been permanently placed in a storage warehouse in this city. The Secretary would urge any members who desire to obtain complete sets to secure them without delay, as there are but few copies left of the earlier years, and the edition for 1902 was entirely destroyed by fire.

All the Sections have had meetings during the year which have been unexcelled in point of attendance and character of work done. No essays have been received by the Council in competition for the several prizes offered. It is to be hoped that during the coming year many of the members will enter into this competition, and, especially, that some will feel disposed to work for the research fund.

In accordance with the vote of the Society at the last meeting, the Council has made a revision of the Constitution and By-laws, a copy of which you have received and which will, in due course, be presented to you for consideration.

There have been no resignations during the year. Seven have been dropped for non-payment of dues. The present membership, including those elected at this meeting, is 281, of which number twenty-two are non-residents.

THOMAS J. HARRIS,

Secretary.

Treasurer's Report, May 31, '06, to May 31, '07.

RECEIPTS.

Balance from 1905-1906	$2,613.66
Fourteen initiation fees at $5.00	70.00
190½ current dues at $10.00	1,905.00
35 back dues at $10.00	350.00
1 back due at $5.00	5.00
Transactions	42.44
Total cash for year	$4,986.10

ACCOUNTS DUE THE SOCIETY.

Members in arrears for one year, 61 at $10.00	$ 610.00
Members in arrears for two years, 23 at $10.00	230.00
Total outstanding accounts	$ 840.00

SAVINGS ACCOUNTS.

Two 1,000 bonds, 6 per cent coupons, present value	$2,100.00
Dividends in Lincoln Savings Bank	60.00
Total	$2,160.00

DISBURSEMENTS.

Reporting Kansas City meeting	$ 215.00	
		$ 215.00
Secretary's expenses, '05-'06	225.83	
Secretary's expenses, '06-'07	140.37	
		366.20
Two bonds	2,120.00	
		2,120.00
Manhattan Storage	67.80	
		67.80
Printing and Postage	76.74	
Clerical work	21.21	
Engrossing certificates	6.00	
		103.95
Stationery	47.50	
		47.50
Indexing (Library Committee)	100.00	
		100.00

Deficit on Annual Dinner	65.00	
		65.00
Section Meetings—		
Western Section	50.00	
Eastern Section	25.00	
		75.00
Transactions, 1905	446.00	
		446.00
Protested check	11.29	
		11.29
Treasurer's Office—		
Miss Stuart	50.00	
		50.00
Total expenses for 1906-1907		$3,667.74

E. W. Day, Treas.

May 31st, 1907.

The above account audited and found correct.

Otto J. Stein,
Auditing Committee.

The Auditing Committee reported that the Treasurer's report had been found to be correct. On motion this report was accepted, placed on file, and the committee discharged.

The Nominating Committee reported as follows:

President—E. W. Day, Pittsburgh.

Secretary—T. J. Harris, New York.

Treasurer—C. Q. Jackson, Pittsburgh.

Vice-Presidents—

Chairman Eastern Section—E. N. Crockett, M. D., Boston, Mass.

Chairman Middle Section—J. F. Barnhill, M. D., Indianapolis, Ind.

Chairman Western Section—Wm. H. Roberts, M. D., Pasadena, Cal.

Chairman Southern Section—H. O. Reik, M. D., Baltimore, Md.

Council—Wendell C. Phillips, Jas. E. Logan, Fred C. Cobb, Jas. F. McKernon, H. W. Loeb, Chas. W. Richardson, D. Braden Kyle, J. F. McCaw, C. R. Holmes.

Library Committee—H. Holbrook Curtis, Phillip D. Kerrison, Lewis A. Coffin, David G. Yates, John B. Rae.

Publication Committee—Wm. H. Haskins, John D. Richards, Otto J. Stein.

Report of Publication Committee.

To the Council,

American Laryngological, Rhinological and Otological Society.

Gentlemen:

The Publication Committee has the honor to submit the following report:

The Transactions for the year 1906, the largest in the history of the Society, were finally issued on May 1, 1907, after many difficulties, the chief of which were the getting of the papers from the authors, and the inexcusable delay in returning the various discussions. We desire most urgently to request that in the future the authors be thoughtful enough carefully to prepare the papers submitted. The great majority of them are invariably found to be full of errors, both typographical and otherwise. If published exactly as received, they would bring great discredit upon the Society. We do not feel that the Publication Committee should be called upon to make these papers readable, as is often the case, but that the authors should send in their papers ready for the printer. The committee would then merely have to see that the printer published them exactly as written. Many weary hours have to be spent in preparing the manuscripts, as can be shown by examining the papers submitted, which should be unnecessary, and which is, frankly speaking, a great imposition on the members who are shouldered with the responsibility of publishing them, and upon whom the blame must fall.

Respectfully submitted.

Chevalier Jackson,
J. D. Richards,
W. H. Haskin,
Publication Committee.

Report of Committee on Society Journal.

Dr. Bert Ellis, Chairman, of Los Angeles, Cal., reported that the Committee had investigated the matter of the publication of a journal to be known as the official organ of the Society. While it was desirable to publish such a journal, the committee believed it would not be a paying investment. To publish a journal of 100 pages would cost $2,400 per year, for printing, mailing, etc. It would be necessary for two men to give a good deal of time to it gratis. It would not be possible for some years to publish more than 1,000 copies, which would not be a good investment for advertisers.

Report of Committee on the President's Address.

Mr. President: Your committee appointed to consider the recommendations made by the President in his annual address, beg to report that we have carefully considered the appointment of a committee for the purpose of perfecting an official nomenclature of the names of diseases and operations, used in Laryngology, Rhinology and Otology. It is to be hoped that this committee will be able to present at the next annual meeting, for the consideration of this Society, a scientific and simplified nomenclature, stripped of the multiple and often incorrect or meaningless synonyms, which in the past have, barnacle like, fastened themselves to so many diseases and operations. We realize that this is no easy task, and that to become effectual, we should have the co-operation of the other leading societies of our specialty.

In order to carry out this work to the best advantage, we believe that the subject should be divided into three parts, i. e., Ear, Nose, and Throat, and the men selected should be so located that they can easily meet for conferences during the coming year. We recommend that this committee be composed of Dr. A. Coolidge, of Boston, who will take Rhinology; Dr. James F. McKernon, of New York, Otology, and Dr. D. Braden Kyle, of Philadelphia, Laryngology.

Secondly—In regard to this Society establishing a journal devoted to our specialty, we feel that this is a very meritorious question, but also one that demands most careful consideration, and believe that such an undertaking would be more likely to culminate successfully if said journal were to become the official organ of all leading societies devoted to our specialty. With this end in view,

we recommend the following committee: Dr. E. Fletcher Ingalls, Dr. Edward B. Dench and Dr. Charles W. Richardson. Said committee to make its report at our next annual meeting.

Respectfully submitted.

E. Fletcher Ingalls,
Frank B. Sprague,
D. Braden Kyle,
James E. Logan,
Christian R. Holmes.

It was moved, seconded and carried that the recommendations of the committee be adopted by the Society.

The recommendations of the Council on the Revision of the Constitution and By-Laws were unanimously approved, and the Constitution and By-Laws adopted as recommended.

At the executive session of the third day the officers named by the Nominating Committee were unanimously elected. The President, Dr. Phillips, appointed a committee to escort the President-elect, Dr. E. W. Day, of Pittsburg, to the Chair.

It was moved, seconded and carried unanimously that a vote of thanks be offered to the Trustees of the New York Academy of Medicine for the courtesy of the use of the building, and that the Secretary be authorized to transmit the resolutions to the Trustees.

It was also moved, seconded and carried that a vote of thanks be offered to the officers of the Laryngological, Rhinological and Otological Sections of the Academy, and to the local members, for the excellent manner in which they have cared for the Society socially, as well as in a scientific way.

It was moved, seconded, and carried by a rising vote, that it be the sense of this meeting that the Journal selected for the publication of the Transactions of this Society should be given the first right of publication of all papers and discussions presented before this organization.

On motion, a vote of thanks was offered to Professor Killian for the honor of his presence, and for his excellent contributions to the scientific program.

FELLOWS.

Abraham, Jos. H.....616 Madison Ave., New York
Abrams, Alva E.....78 High St., Hartford, Conn.
Adams, John L.....38 East 51st St. New York
Alderton, Henry A. .....142 Clinton St., Brooklyn, N. Y.
Allport, Frank A.....92 State St., Chicago, Ill.
Anderson, Willis S.....606 Washington Arcade, Detroit, Mich.
Ard, F. C.....604 Park Ave., Plainfield, N. J.
Arnold, J. Dennis.....1296 Van Ness Ave., San Francisco, Cal
Arrowsmith, Hubert.....180 Clinton St., Brooklyn, N. Y.
Atkinson, Wm. J.....111 North Main St., Paterson, N. J.
Babbitt, Jas. A.....112 South 18th St., Philadelphia, Pa.
Ballenger, Wm. L.....100 State St., Chicago, Ill.
Bane, Wm. C.....Commonwealth Bldg., Denver, Colo.
Barnhill, John F.....Indianapolis, Ind.
Battle, S. Westray.....13 Church St., Asheville, N. C.
Beck, Jos.....100 State St., Chicago, Ill.
Berens, Bernard.....2041 Chestnut St., Philadelphia, Pa.
Berens, T. Passmore.....35 Park Ave., New York
Birkett, H. S.....252 Mountain St., Montreal, Can.
Blake, Clarence J. .....226 Marlborough St., Boston, Mass.
Booth, Burton S.....21 First St., Troy, N. Y.
Borden, Charles R. C.....223 Newberry St., Boston, Mass.
Braislin, W. C.....556 Washington Ave., Brooklyn, N. Y.
Brown, J. Price.....37 Carlton St., Toronto, Ont., Can.
Bryant, Wm. S.....57 West 53d St., New York
Bulette, Wilbur W.....Fifth and Santa Fe Aves., Pueblo, Colo.
Burnett, Peter V.....170 Keap St., Brooklyn, N. Y.
Calhoun, A. W.....62 Marietta St., Atlanta, Ga.
Campbell, Wm. E.....606 Century Building, Atlanta, Ga.
Canfield, R. Bishop.....Ann Harbor, Mich.
Chambers, Talbot R.....490 Jersey Ave., Jersey City, N. J.
Cheatham, Wm.....303 West Chestnut St., Louisville, Ky.
Chenery, Wm. E.....222 Huntington Ave., Boston, Mass.
Chisholm, W. Alexander .....236 W. 44th St., New York
Christy, T. C.....717 Westinghouse Building, Pittsburg, Pa.
Clark, Jos. Payson.....409 Marlboro St., Boston, Mass.

Clemens, Jas. B.....15 East 48th St., New York
Cline, L. C. .....224 North Meridan St., Indianapolis, Ind.
Coakley, C. G.....53 West 56th St., New York
Coates, Geo. M.....334 S. 19th St., Philadelphia, Pa.
Cobb, Frederick C.....11 Marlboro St., Boston, Mass.
Coffin, Lewis A.....156 West 58th St., New York
Coggeshall, Henry.....40 East 58th St., New York
Cohen, J. Solis.....1824 Chestnut St., Philadelphia, Pa.
Collins, Burnett.....645 St. Marks Place, Brooklyn, N. Y.
Connell, J. C. .....265 King St., Kingston, Ont., Can.
Corwin, Theodore.....5 West Park St., Newark, N. J.
Coolidge, A.....613 Beacon St., Boston, Mass.
Cott, Geo. F. .....85 North Pearl St., Buffalo, N. Y.
Cox, Chas. N.....257 Jefferson Ave., Brooklyn, N. Y.
Craig, Robert H.....186 Peel St., Montreal, Can.
Crockett, Eugene A.....298 Marlboro St., Boston, Mass.
Culbert, Wm. Ledlie.....54 East 34th St., New York
Culp, John F.....Harrisburg, Pa.
Curtis, H. Holbrook.....118 Madison Ave., New York
Day, Ewing W.....Westinghouse Building, Pittsburg, Pa.
Daly, Walter S.....Ogdensburg, N. Y.
Dench, Edward B. .....17 West 46th St., New York
Dessar, L. A. .....156 West 58th St., New York
Donnellan, Patrick S.....1028 Spruce St., Philadelphia, Pa.
Dudley, Wm. F.....32 Livingston St., Brooklyn, N. Y.
Dudley, Wm. H.....Los Angeles, Cal.
Duel, Arthur B.....254 Madison Ave., New York
Dunn, John.....314 East Franklin St., Richmond, Va.
Dye, Hobart S.....1404 L St., N. W., Washington, D. C.
Eagleton, Wells P.....15 Lombardy St., Newark, N. J.
Ellis, H. Bert.....245 Bradbury Building, Los Angeles, Cal.
Emerson, Francis P.....657 Boylston St., Boston, Mass.
Emerson, Linn .....Orange, N. J.
Farrell, Thomas H.....236 Genessee St., Utica, N. Y.
Fetterolf, Geo.....328 South 15th St., Philadelphia, Pa.
Fitzpatrick, T. V.....26 Garfield Place, Cincinnati, Ohio
Fleming, Ernest W.....214 Bradbury Block, Los Angeles, Cal.
Foster, John M.....11 Stedman Bldg., Denver, Colo.
Freeman, Walter J.....1720 Locust St., Philadelphia, Pa.
Freudenthal, Wolff.....1003 Madison Ave. New York
Fryer, Blencowe, E. ....Cor. 9th and Cherry Sts., Kansas City, Mo.

Gallaher, Thomas J.....California Building, Denver, Colo.
Gibb, Jos. S.....1907 Chestnut St., Philadelphia, Pa.
Gildea, Patrick F.....2 Cascade Ave., Colorado Springs, Colo.
Goff, Waldo P.....Clarksburg, W. Va.
Goldsmith, Perry G.....Belleville, Ont.
Goldstein, Max A.....3858 Westminster Place, St. Louis, Mo.
Gradle, Henry.....100 State St., Chicago, Ill.
Graef, Charles.....1076 Boston Road, New York
Grayson, Chas. P.....251 South 16th St., Philadelphia, Pa.
Greene, D. C., Jr.....12 West Cedar St., Boston, Mass.
Griffin, Edwin H.....112 West 45th St., New York
Gruening, Emil.....35 East 57th St., New York
Haddock, Chas. W.....14 Bartlett St., Beverly, Mass.
Hall, D. Walton.....Bryant Building, Kansas City, Mo.
Halsted, Thomas H. .....831 University Block, Syracuse, N. Y.
Harris, Thomas J.....117 East 40th St., New York
Hartz, Henry J.....27 Adams St., East Detroit, Mich.
Haskin, Wm. H.....42 East 41st St., New York
Hastings, Hill .....Los Angeles, Cal.
Hedges, Halsted S.....Charlottesville, Va.
Held, R. Johnson.....616 Madison Ave., New York
Hepburn, Neill J. .....317 West 23d St., New York
Hinkel, F. W.....412 Franklin St., Buffalo, N. Y.
Holmes, Christian R.....8 East 8th St., Cincinnati, Ohio
Holmes, Edgar M.....531 Beacon St., Boston, Mass.
Hopkins, F. E. .....25 Harrison Ave., Springfield, Mass.
Hopkins, Wm. E.....803 Sutter St., San Francisco, Cal.
Hubbard, Dwight I.....135 West 94th St., New York
Hubbard, Thomas.....205 Ontario St., Toledo, Ohio
Hurd, Lee M.....15 East 48th St., New York
Hyatt, Frank.....1022 14th St., N. W., Washington, D. C.
Ingals, E. Fletcher.....34 Washington St., Chicago, Ill.
Ingersoll, John M.....50 Euclid Ave., Cleveland, Ohio
Jack, F. L.....215 Beacon St., Boston, Mass.
Jackson, Chevalier.....428 Park Building, Pittsburg, Pa
Jackson, Oliver H.....34 North Main St., Fall River, Mass.
Joachim, Otto.....501 Marecha Building, New Orleans, La.
Jones, Clarence Porter .....Newport News, Va.
Keiper, Geo. F.....Lafayette, Ind.
Kenefick, Jos. A.....78 E. 54th St., New York
Kennon, Beverley R.....64 Granby St., Norfolk, Va.

Kent, J. L.....1101 Church St., Lynchburg, Va.
Kerrison, Phillip D.....58 West 56th St., New York
Kimball, Irving E.....717 Congress St., Portland, Me.
Kirkendall, J. S.....Ithaca, N. Y.
Kirkpatrick, E. A.....Pleasant St., Halifax, N. S.
Knapp, Arnold.....26 W. 40th St., New York
Knowles, W. F.....220 Clarendon St., Boston, Mass.
Kopetsky, Samuel J.....616 Madison Ave., New York
Koyle, F. H.....199 Main St., Hornellsville, N. Y.
Kuyk, D. A.....Richmond, Va.
Kyle, D. B.....1517 Walnut St., Philadelphia, Pa.
Kyle, John J.....The Newton Claypool Building, Indianapolis, Ind.
Lederman, M. D.....58 East 75th St., New York
Leland, Geo. A.....354 Commonwealth Ave., Boston, Mass.
Leonard, Z. L.....19 West 120th St., New York
Lester, John C.....179 Schermerhorn St., Brooklyn, N. Y.
Levy, Robert.....300 California Building, Denver, Colo.
Lewis, Robert, Jr.....48 West 40th St., New York
Lincoln, Wm. R.....Lenox Building, Cleveland, Ohio
Linhart, C. P.....Hotel Vendome, Columbus, Ohio
Loeb, H. W.....537 North Grand Ave., St. Louis, Mo.
Logan, Jas. E.....1208 Wyandotte St., Kansas City, Mo.
Lowman, John B.....142 Park Place, Johnstown, Pa.
Lutz, Stephen H.....284 Hancock St., Brooklyn, N. Y.
Mabon, John S.....721 Sandusky St., Allegheny, Pa.
Macpherson, D.....467 Manhattan Ave., New York
Makuen, G. Hudson.....1627 Walnut St., Philadelphia, Pa.
Manson, Charles A.....2 West 94th St., New York
Mason, William Beverly..812 Connecticut Ave., Washington, D. C.
Means, Chas. S.....715 North High St., Columbus, Ohio.
Mial, Leonidas L.....133 West 11th St., New York
Miller, Frank E.....22 West 31st St., New York
Miller, Lewis H.....287 Clinton Ave., Brooklyn, N. Y.
Milligan, Robert.....Smith Block, 6th and Liberty, Pittsburg, Pa.
Mooney, Jas. J.....393 7th St., Buffalo, N. Y.
Moore, Thomas W.....1048 Third Ave., Huntington, W. Va.
Morgenthau, Geo.....34 Washington St., Chicago, Ill.
Mosher, Harris P.....828 Beacon St., Boston, Mass.
Moss, Robert E.....Hicks Bldg., San Antonio, Tex.
Mullins, John B.....1516 H St., Washington, D. C.
Munger, Carl E.....Waterbury, Conn.

Murphy, John W.....4 W. 17th St., Cincinnati, Ohio
Murray, Gilbert D.....436 Wyoming Ave., Scranton, Pa.
Myers, Henry L.....151 Granby St., Norfolk, Va.
Myles, Robert C.....46 West 38th St., New York
McCaw, James F.....35 Washington St., Watertown, N. Y.
McClelland, Lefferts A.....78 McDonough St., Brooklyn, N. Y.
McCoy, John J.....139 West 97th St., New York
McCullaugh, Samuel.....35 Park Ave., New York
McGahan, Chas. F.....Aiken, S. C.
McKernon, Jas. F.....62 West 52d St., New York
McKimmie, O. A. M...1330 Mass. Ave., N. W., Washington, D. C.
McLaury, Frank H.....224 West 42d St., New York
McReynolds, John O.....Frost Building, Dallas, Texas
Norris, C. W.....Lexington, Ky.
Off, Henry J.....121 South 18th St., Philadelphia, Pa.
O'Kelly, Jas. P.....501 Macheca Building, New Orleans, La.
Oppenheimer, Seymour.....45 East 60th St., New York
Packard, Francis R.....19th and Pine Sts., Philadelphia, Pa.
Park, J. Walter.....32 North 2d St., Harrisburg, Pa.
Parker, Edward F.....70 Hassell St., Charleston, S. C.
Patterson, Jas. A...805 North Trejon St., Colorado Springs, Colo.
Payne, Redmond W.....135 Geary St., San Francisco, Cal.
Peete, C. H.....Macon, Ga.
Phillips, Wendell C.....40 West 47th St., New York
Pierce, Norval H.....31 Washington St., Chicago, Ill.
Pischel, Kaspar .....San Francisco, Cal.
Powell, Stephen C.....Newport, R. I.
Quinlan, F. J.....33 West 38th St., New York
Rae, John B.....616 Madison Ave, New York
Ray, J. M. .....432 West Chestnut St., Louisville, Ky.
Reik, Henry O.....412 Cathedral St., Baltimore, Md.
Renner, W. Scott.....361 Pearl St., Buffalo, N. Y.
Reuling, Geo.....103 West Monument St., Baltimore, Md.
Richards, George L. .....84 North Main St., Fall River, Mass.
Richards, John D.....44 West 49th St., New York
Richardson, Chas. W....1317 Connecticut Ave., Washington, D. C.
Roberts, Walter.....33 South 19th St., Philadelphia, Pa.
Roberts, W. Humes.....Pasadena, Cal.
Robinson, John A.....209 W. 85th St., New York
Roe, John O.....28 Clinton Ave., N., Rochester, N. Y.
Rogers, Frederick T.....117 Broad St., Providence, R. I.

Roosa, D. B. St. John.....20 East 30th St., New York
Root, Arthur G.....218 State St., Albany, N. Y.
Ross, Geo. T.....945 Dorchester St., Montreal, Can.
Roy, Dunbar.....Grand Opera House Block, Atlanta, Ga.
Sandles, C. C.....Westinghouse Building, Pittsburg, Pa.
Sauer, Wm. E.....Grand and Washington Aves., St. Louis, Mo.
Schadle, J. E.....140 Lowry Arcade, St. Paul, Minn.
Seiss, Ralph W.....213 South 17th St., Philadelphia, Pa.
Sharp, J. Clarence.....62 West 46th St., New York
Shattuck, Warren S., Jr. .....147 Clinton St., Brooklyn, N. Y.
Sheppard, J. E.....130 Montague St., Brooklyn, N. Y.
Shorter, J. H.....Macon, Ga.
Shields, Wm. B.....303 North Grand Ave., St. Louis, Mo.
Shurly, B. R.....32 W. Adams Ave., Detroit, Mich.
Smith, Harmon.....44 West 49th St., New York
Smith, S. Mac Cuen.....1700 Walnut St., Philadelphia, Pa.
Smith, W. Harvey.....250 Donald St., Winnipeg, Manit.
Smith, E. Terry.....75 Pratt St., Hartford, Conn.
Smyth, Herbert E.....376 John St., Bridgeport, Conn
Snow, Sargent F.....707 University Block, Syracuse, N. Y.
Solenberger, Amos R..106 E. St. Vrain Ct., Colorado Springs, Colo.
Sondern, Paul F.....52 South Fitzhugh St., Rochester, N. Y.
Sprague, Frank B.....27 Stewart St., Providence, R. I.
Spratt, Charles Nelson, 310 Syndicate Arcade, Minneapolis, Minn.
Steel, Geo. E.....124 West 119th St., New York
Stein, Otto Jacob.....100 State St., Chicago, Ill.
Sterrett, John Kennedy.....320 Smith Block, Pittsburg, Pa.
Stillman, F. L.....118 East Broad St., Columbus, Ohio
Stout, Geo. C.....34 South 18th St., Philadelphia, Pa.
Stucky, J. A.....The McClelland Building, Lexington, Ky.
Theisen, Clement F.....172 Washington Ave., Albany, N. Y.
Thigpen, C. A.....13 South Ferry St., Montgomery, Ala.
Thigpen, F. M.....Pensacola, Fla.
Thompson, John A.....628 Elm St., Cincinnati, Ohio
Thompson, John J.....40 W. 47th St., New York
Thompson, J. C.....202 Ninth Ave., Pittsburg, Pa.
Thrasher, A. B.....N. E. Cor. 7th and Race Sts., Cincinnati, Ohio
Thrasher, Wade.....N. E. Cor. 7th and Race Sts., Cincinnati, Ohio
Trowbridge, D. H.....Fresno, Cal.
Turnbull, Thomas.....835 Wheaton Ave., Allegheny, Pa.
Tyson, Henry Hawkins.....47 West 51st St., New York

Vail, Derrick T.....24 East 8th St., Cincinnati, Ohio
Vansant, Eugene L.....1929 Chestnut St., Philadelphia, Pa.
Voislawsky, A. P.....128 West 59th St., New York
Voorhees, Sherman.....408 North Main St., Elmira, N. Y.
Wagner, Henry L.....506 Sutter St., San Francisco, Cal.
Wales, Ernest DeW.....320 N. Meridian St., Indianapolis, Ind.
Waterman, Jas. S.....676 St. Marks Ave., Brooklyn, N. Y.
Webster, Geo. A.....419 Boylston St., Boston, Mass.
Wells, Walter A.....1113 14th St., Washington, D. C.
White, Joseph A.....200 East Franklin St., Richmond, Va.
Wilson, Norton L.....410 Westminster Ave., Elizabeth, N. J.
Winslow, John R.....114 Franklin St., Baltimore, Md.
Wishart, D. J. Gibb.....47 Grosvenor St., Toronto, Can.
Wood, George B.....129 South 18th St., Philadelphia, Pa.
Woodward, John F.....Norfolk, Va.
Yates, D. G.....79 West 104th St., New York

HONORARY FELLOWS.

Ballance, C. A., London, England.
Gradenigo, G., Turin, Italy.
Grant, Dundas, London, England.
Heryng, T., Warsaw, Russia.
Joel, J., Paris, France.
Karwowski, Constantine, Warsaw, Russia.
Killian, G., Freiburg, Germany.
Law, Edward, London, England.
Luc, H., Paris, France.
Macintyre, John, Glasgow, Scotland.
Martin, A., Paris, France.
Moure, E. J., Bordeaux, France.
Martinez, Emilio, Havana, Cuba.
Onodi, Adolph, Budapest, Hungary.
Politzer, A., Vienna, Austria.
Poyet, George, Paris France.
Semon, Sir Felix, London, England.
Schmiegelow, E., Copenhagen, Denmark.
Sendziak, J., Warsaw, Russia.
Turner, Logan, Edinburgh, Scotland.
Woakes, W., London, W., England.
Wolfenden, R. Norris, London, England.
Ziem, C. H., Danzig, Germany.

RF
1
A53
1907
Biological
& Medical
Serials

American Laryngological, Rhinological and Otological Society
Transactions

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY

STORAGE